Food Patterns, Nutrient Intakes and Metabolic Demand in Children of Short Stature, Children of Short Stature Receiving Growth Hormone Treatment and Normal Height Children

A thesis presented for the degree of
Master of Philosophy

by

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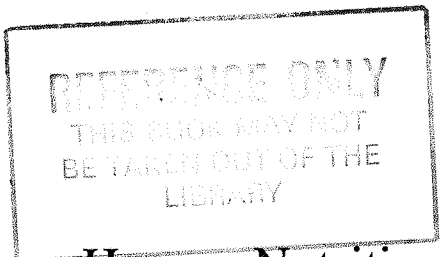

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ABSTRACT

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FOOD PATTERNS, NUTRIENT INTAKES AND METABOLIC DEMAND IN CHILDREN OF SHORT STATURE, CHILDREN OF SHORT STATURE RECEIVING GROWTH HORMONE TREATMENT AND NORMAL HEIGHT CHILDREN

By Catherine Mary Smith

Short stature is a condition which is clinically investigated especially when seen in children. If the reason for the short stature cannot be diagnosed then growth hormone therapy becomes a possible treatment.

In this thesis 3 groups of 9-10 year old children were taken with varying heights. The first group were short in stature, the second were short in stature and having growth hormone treatment and the third group were children of normal height. Aspects of energy metabolism were investigated in 3 ways - the pattern of food consumption which provided this energy, the amount of energy and other nutrients consumed in the diet and whether this energy satisfied metabolic demand.

The short children consumed an increased quantity of high fat, high sugar foods to provide for their energy, but had diets of similar nutrient density. Metabolic demand and dietary energy intake in both groups of short children were increased in comparison to the normal height children when expressed in terms of body size. When expressed in absolute amounts the short children have a decreased metabolic demand and dietary energy intake. Body size is an important parameter to use as a reference standard rather than age. Body size should be considered when expressing energy in the form of metabolic demand and dietary intake.

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MAIN ABBREVIATIONS USED IN THE TEXT

DNA	Deoxyribonucleic acid
BMR	Basal Metabolic Rate
LBM	lean body mass
MJ	megajoules
kJ	kilojoules
kJ/kg/LBM	kilojoules per kilogramme of lean body mass
RQ	respiratory quotient
%	percentage
DRV	Dietary Reference Value
EAR	Estimated Average Requirement
RNI	Reference Nutrient Intake
LRNI	Lower Reference Nutrient Intake
kg	kilogramme
g	gramme
mg	milligramme
µg	microgramme
kJ/g	kilojoule per gramme
IGF	insulin-like growth factor
rhGh	recombinant growth hormone
Iu/m^2/week	International units per metre squared per week
°C	degrees centigrade
v	volt
SPSS	Statistical Package for Social Scientist
d	day
SDS	Standard deviation score
yrs	years
cms	centimetres

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"We the clay, You the potter,

we are all the work of Your hand."

Isaiah ch 64,v 8.

INTRODUCTION

Through the school years, growth is monitored by measuring height and body weight. These values when compared to growth standards can give a health professional an indication of how well or how poorly a child is growing. The pattern of growth is important in assigning a child to a diagnostic category. If the height and weight of a child begins to plateau then this will alert the health professional to investigate the cause. Likewise, monitoring growth will also give reassurance that a child is growing as expected for age.

When a child fails to develop an increase in height or weight, or when the rate of increase begins to slow according to their chronological age, the child becomes small in stature. By being shorter or smaller in body size the child can be affected psycho-socially as well as physically.

Short stature children can often come against a variety of stresses unbeknown to themselves. That of social isolation, low self-esteem, conflicts over body image and a sense of being weak and incompetent. Since these children can be excluded on a basis of size, they often do not participate in the full range of peer activities. Short children may only cope with this by excelling in an independent skill; for example music, theatre, non-team sports and intellectual pursuits.

Diet is a major determinant of how well a child will grow. On a global scale, malnutrition is the most common cause of growth failure with an estimated two thirds of the worlds children undernourished. The nutritional supply must

satisfy the metabolic demand for energy in order for growth to occur. The metabolic demand for this energy arises from several components, the largest single component being Basal Metabolic Rate (BMR). If this energy demand is not met then poor growth will occur and the child will become short in stature.

In our Western society today, increasing demand is placed on children to be of 'normal' height. However, there is increased pressure for boys rather than girls to be tall and consequently a higher proportion of small boys are referred for growth evaluation than are small girls.

The extent to which the short child is disadvantaged and failing to reach their potential, will decide whether a growth promoting treatment such as growth hormone is selected for use.

Since 1985 when the first biosynthetic growth hormone preparation was granted a product licence, supplies are now more widely available. Because of the availability and flexibility of its use, many children are now receiving growth hormone treatment.

Growth hormone therapy will increase height at higher rates than normal in an effort to 'catch up'. The treatment is known to have lipolytic and anabolic effects on body composition. This in turn must increase the metabolic demand and the possible use of more energy being for BMR and growth. Along with this, dietary intake will therefore play an even more important role in maintaining adequate levels of substrate needed for the child's growth and development.

This thesis specifically addresses the relationship between diet and growth in short stature children and the

effect that accelerated growth achieved through growth hormone treatment may have on food intake.

CHAPTER 1

1. REVIEW OF THE LITERATURE

Practising clinicians and dietitians face a fundamental challenge in assessing the adequacy of a child's diet, especially if that child has disease or appears to be growing poorly. This review explores the literature that surrounds children's dietary intakes, their growth and metabolic requirements.

The first section of the review considers diet, in particular the dietary intakes of children. The focus is on energy intake and other nutrients. Also discussed is how dietary intake is measured and the factors that alter dietary food patterns and nutrient intakes.

The second section of the review discusses aspects of growth and the relationship that growth has with nutritional status.

In the final section, short stature is reviewed, in particular the determinants and consequences of being short in stature. The use of growth hormone in the treatment of short stature is explored, in particular the impact it may have on dietary intake.

1.1 DIETARY INTAKE

Diet is a major determinant of how well a child will grow. The nutritional supply must satisfy the metabolic demand for energy, in order for growth to occur.

1.1.1 DEFINING DIETARY INTAKE

A diet has a food pattern or a food frequency which are the foods or mixtures of foods that are regularly consumed by an individual. Dietary intake can be described as the food that a person regularly consumes. Food is any solid or liquid which when swallowed can supply energy from which the body can produce movement, material and substrates necessary to regulate energy production and processes of growth and repair. The components of foods which have these functions are nutrients.

1.1.2 MEASUREMENT OF DIETARY INTAKE

The measurement of dietary intake is the main component to any nutrition study and there are various methods that can be used [Bingham 1987]. All the methods described below are available in dietetic practice and useful tools in the assessment of dietary intake, with each one being suited to different types of study.

Food records can firstly be measured by a weighed food intake method where all food and drink is measured using accurate weighing scales for a period of up to 7 days, but can be longer. Secondly, the 24 hour recall method can be used, where estimates of food over the last 24 hour period are recalled. Another method known as 'diet history' incorporates a 24 hour recall but with further questions on quantities and frequencies of intake of particular foods taken over a longer period of time. The last method is the

food frequency questionnaire which can be used. Here, the interviewer asks how often foods are eaten with a range of various frequencies of consumption. The questionnaire may be semi-quantitative using photographs or models of foods.

If the weighed food intake method is used and kept for any less time than 7 days then errors of the individual daily intake and the estimation of metabolisable energy intake using food tables becomes more apparent. Food intakes are connected to nutrient intakes with the use of food consumption tables. The results of a dietary survey depend ultimately on the quality of food tables. Accuracy obtained depends on the food sampling procedure and methods of analysis. In the case of energy and other nutrients of certain foods in the tables there are incomplete values indicating the analysis was not as complete as for some of the other foods. Other inaccuracies can occur with the human error of coding foods.

For the purpose of comparison with other physiological characteristics for example, metabolic rate or blood pressure; a 7 day weighed food record is probably the minimum reliable period for the assessment of energy intake. If other nutrients are to be studied, the period of observation might need to be longer. Randomly picked days are not sufficient as this may result in a reduction of food intake just for that day and if the time of recording is more than 7 days, it is likely not to be tolerated. Preferably the length of time should be divided into 3-4 day periods over the course of a few months.

Self-reporting of dietary intake is subject to inaccuracies. Some of these inaccuracies arise from

subjects incomplete record keeping and unrepresentative food selection. The error in coding and translation of foods into nutrients can also affect results.

It is well known that self-reporting of dietary intake in obese subjects is always much lower than the intake actually is in everyday life [Garrow 1978]. As the degree of under-reporting increases with intake, it is speculated that individuals tend to report intakes that are closer to perceived norms than to actual intakes.

Measurement error in dietary intakes of children appears to increase as the child becomes older and moves into adolescence [Livingstone 1992]. Up until the age that a child moves into adolescence they rely on their parent or carer for the provision of food. This makes the dietary recording the responsibility of the parent. Once into adolescence with the increased independence, it becomes more likely that food is consumed out of the home and not reliably weighed or recorded.

1.1.3 VALIDITY OF DIETARY INTAKE MEASUREMENTS

Validity of measurements can be described as

(i) 'a reasonable representation of the true situation, for example if a study was repeated in the same population using the same methods, approximately the same results would be obtained' [Margetts 1991]

(ii) 'The measure of exposure or outcome of a study actually measuring that exposure or outcome' [Margetts 1991].

The validity of a dietary study should measure true food intake but no common standard exists. There have been attempts to validate dietary intake by comparing energy intake against expenditure either as a multiple of BMR

[Goldberg 1991] or as total energy expenditure using the doubly labelled water technique [Coward 1988, Livingstone 1990]. Alternatively a validation for specific nutrients has been proposed. For example, protein intake validated by urinary nitrogen excretion, sodium intake by urinary sodium excretion [Bingham 1987].

These two validity techniques do not validate other major nutrients and micro-nutrients but if energy and protein measures compare well to intakes recorded, then it can be assumed that other nutrient intakes have been reported with the same degree of accuracy since other nutrients cannnot be altered without changes to energy and protein intakes.

However, like all other techniques of measurement, with sufficient skill and care by the recorder and the researcher, reliable results can be obtained using the seven day weighed food intake [Bingham 1987]. It is important to recognise that data reported in the literature is rarely supported by evidence of validity. If the same method of assessment is used to compare dietary intakes between groups then internal validity can be assumed as any errors occurring are likely to be similar across the groups. The assumption may not be so if the comparing groups eat substantially differently. However, groups consuming a similar mixed diet are less likely to have errors occurring.

Ideally to validate dietary records, the same or different dietary techniques should be repeated. For example, if the energy intake was high in a weighed inventory and also with a food frequency questionnaire then it is likely that the 'ranking order' provides consistency with the reporting.

1.1.4 REFERENCE POINTS OF ADEQUACY

Over the last 30 years in the U.K., 'standards' in the form of Recommended Intakes for Nutrients and Recommended Daily Amounts have existed. The Department of Health and Social Security in 1969 [DHSS 1969] published this report recommending the daily energy and nutrient intakes for various groups of the population. In 1979, the Recommended Daily Intakes were updated to the Recommended Daily Amounts [DHSS 1979]. These recommendations for nutrients were designed to ensure that the needs of almost all healthy persons were covered. The recommendations for energy for each group of people was set at the estimated average requirement, half of the individuals in the group requiring more and half less.

The aim of a standard is to ensure every individual receives enough of a nutrient to meet their needs. New standards have now been set which clarify the terminology and reduce the chance of the values being used incorrectly by investigators. These are collectively known as the Dietary Reference Values (DRV) [DHSS 1991]. The DRV's have up to four figures set for each nutrient. The adequacy of dietary intakes in groups and individuals can therefore be assessed for those who may have high requirements and those who may have low.

The definitions of the values are as below:

ESTIMATED AVERAGE REQUIREMENT (E.A.R) - estimate of the average requirement or need for food, energy or nutrient.

REFERENCE NUTRIENT INTAKE (R.N.I) - an amount of a nutrient that is enough for almost every individual, at least 97% of the population. It covers those who have high needs for the nutrient. If individuals are consuming the RNI of a nutrient, they are most unlikely to be deficient in that nutrient.

LOWER REFERENCE NUTRIENT INTAKE (L.R.N.I) - the amount of a nutrient that is enough for only the small number of people with low needs. If individuals are habitually eating less than the LRNI they will almost certainly be deficient.

The above values are nutritional standards against which groups of individuals can be compared.

The comparison of the reported energy intake against the BMR of the individual may be used as a means of examining the validity of the recorded food intake. Measurements of energy intake which are greater than 1.35 x BMR are likely to be valid [Goldberg 1991].

1.1.5 DIETARY FOOD PATTERNS OF CHILDREN

Throughout childhood there is growth and development during which adequate nutrition is paramount, yet there is little data existing on the food choice in young children. For growth to occur there must be adequate energy to satisfy the needs of metabolic demand and tissue synthesis. This is derived from food in the diet, thus the quantity of energy consumed is important in fulfilling this need.

The adequacy of dietary energy in children remains first

and foremost an important issue regarding growth. In addition to energy, other nutrients such as minerals and vitamins are required to support life at cellular level. Knowing that the energy intake is sufficient does not always mean that other nutrients are adequate. To some extent it can be said that with an increase in energy intake, there will be an increased requirement for all other nutrients. Food types will provide energy but will not always provide the adequate nutrients; for example, sugar is energy dense but contains very few other nutrients.

The reporting of intakes of foods are as important as the reporting of nutrient intakes. Children choose foods to eat and not nutrients. Concern centres on the dietary habits of school children because it has been suggested that the types of foods consumed might be associated with the development of some malignancies and coronary heart disease [McGinnis 1989, Willett 1990a]. Based on the dietary food patterns or quality of the diet it should be possible to provide a prediction of a child's growth and development. Nutrient intakes, particularly energy intake may be the same in two children but depending on the foods providing this energy, growth and size will vary [Allen 1991]. The food pattern and quality of a diet consumed by a child is related to and by be determined by the level of education of the mother [Hagman 1986].

The four studies listed in Table 1.1 report on dietary food patterns in children. Table 1.2 illustrates the mean percentage energy contribution of foods to the total energy intake of children recruited to the studies.

Table.1.1 Authors and subject characteristics of studies reporting food patterns in children.

STUDY	NUMBER, SEX OF CHILDREN	AGE OF CHILDREN	AUTHORS
GLASGOW	419 (198 ♂ / 221 ♀)	14 Years	Durnin et al, 1971
NORTHUMBERLAND	405 (193 ♂ / 212 ♀)	11.5 Years	Hackett et al, 1984#
LONDON	1723 (902 ♂ / 821 ♀)	10-11 Years	DHSS, 1989
NORTHUMBERLAND	379 (184 ♂ / 195 ♀)	11.5 Years	Adamson et al, 1992#

- Three day weighed food diary, all other studies used a 7 day weighed food diary.

Table 1.2 Mean % energy contribution of foods to the total energy intake of children recruited in the Glasgow, London and both Northumberland studies.

STUDIES

	GLASGOW (1971)	NORTHUMBERLAND (1984) Av of	LONDON (1989)	NORTHUMBERLAND (1992)
Meat and meat products (% energy)	14	13	-	16
Milk (% energy)	10	8	7	5
Cakes and biscuits (% energy)	8	9	11 Biscuits only	8
Confectionery, sugar, jam and soft drinks (% energy)	14	14	-	17
Chips and potato (% energy)	10	11.8	8	12
Cereals and bread (% energy)	13	13	10 (Bread only)	8
Crisps (%energy)	-	4	-	4

There were no differences in the mean percentage energy contribution of foods between sexes, so values presented are for boys and girls combined.

In none of these studies was any attempt made to examine the relationship between size and growth with food choice. Ranges were not recorded only means.

Throughout all four studies, meat was the main contributor of energy in the children's diets providing an average 14% of total energy. The next major contribution to energy was confectionery, sugar and soft drinks. In 1971 Durnin reported this contribution being 14% and in 1992 Adamson reported the amount to be 17%, suggestive of rise over the 20 years.

Potatoes and chips were the next major contributor to energy intake, on average 12%. However, in 1971 Durnin reported this contribution to be only 10% thus suggesting a rise over the last 20 years possibly due to the increase in fast food availability providing chips. In addition to the 'fish and chip' shops there are numerous fast food chain outlets all providing quick meals with chips.

In 1971 it appeared from the work of Durnin that bread consumption was higher than reported in 1992 by Adamson, 13% falling to 8%. This suggests that the chip and potato increase have replaced a proportion of bread consumption.

The earlier studies of Durnin 1971, Hackett 1984 and the DHSS 1989 report, give another major contributor to energy to be milk, at an average of 9-10%. Adamson 1992 reports milk consumption as 5%, suggestive of a fall to half.

Cakes and biscuits are being consumed in the same quantities by children today as they were 20 years ago.

Adamson in 1992 reports an intake providing 8% and this remains similar to the work of Durnin in 1971. The DHSS 1989 study however reports biscuits to provide 11% of the total energy.

1.1.6 NUTRIENT INTAKES OF CHILDREN

The quality and quantity of childrens' diets has always been of great concern. There have been various studies investigating the nutritional intakes of British school children. These studies have been listed in Table 1.3. The mean energy and nutrient intakes of the children in these studies are illustrated in Table 1.4

Table 1.3 Authors and subject characteristics of studies reporting on Nutrient intakes in children

STUDY	NUMBER, SEX OF CHILDREN	AGE OF CHILDREN	AUTHORS
GLASGOW	419 (198 ♂ / 221 ♀)	14 Years	Durnin et al, 1971*
KENT	398 (198 ♂ / 190 ♀)	9-11 Years	Cook et al, 1973*
NORTHUMBERLAND	405 (193 ♂ / 212 ♀)	11-15 Years	Hackett et al, 1984#
LONDON	1723 (902 ♂ / 821 ♀)	10-11 Years	DHSS, 1989
DUNDEE	61 (18 ♂ / 43 ♀)	12 Years	McNeill et al, 1991

* - Comparison of nutritional adequacy with Department of Health and Social Security, (DHSS)(1969). All the other studies used comparison of adequacy with DHSS (1979).

- Three day food diary used, all the other studies used 7 day food diaries.

Table 1.4 Mean energy and nutrient intakes in the children recruited in the Glasgow, Kent, Northumberland, London and Dundee studies.

	RNI 11–14 Yr Old ♂	RNI 11–14 Yr Old ♀	GLASGOW (1971)	KENT (1973) ♂	KENT (1973) ♀	NORTHUMBERLAND (1984)	LONDON (1989) ♂	LONDON (1989) ♀	DUNDEE (1991) ♂	DUNDEE (1991) ♀
Energy (MJ)	9.27 (EAR)	7.92 (EAR)	9.6	9.5	8.4	9.4	8.7	7.7	8.9	8.1
Protein (g)	28.3	28.3	70	65	56	64	61	53	61	55
Calcium (mg)	1000	800	908	996	873	896	833	702	822	767
Iron (mg)	11.3	14.8	12.5	11	10	10.5	10	8.6	10	10
Vitamin A (μg)	600	600	–	997	956	698	589	460	494	510
Vitamin C (mg)	35	35	–	49	45	40.4	49.3	49	47	28
Thiamin (mg)	0.4	0.4	–	1.1	0.9	–	1.21	1.03	1.2	1.0
Riboflavin (mg)	1.2	1.1	–	1.6	1.4	–	1.7	1.4	1.5	1.4
Nicotinic acid (mg)	6.6	6.6	–	12	10	–	26.5	23.1	27	24

The Northumberland study [Hackett 1984] and the Glasgow study [Durnin 1971] did not separate boys and girls and so the values are for the sexes combined. These studies did not record ranges only means.

Energy intakes in all the studies when compared to the Dietary Reference Value of Estimated Average Requirement (EAR) [DHSS 1991] are seen to be more than adequate. When each study is compared to the old RDA energy value [DHSS 1969] then energy intakes are below the recommendations. The suggestion has been that the energy reference point set by the DHSS at that time was too high since the children participating in the studies were of normal body size and not suffering signs of inadequate nutrition [Durnin 1971]. It has also been seen that the energy intakes in children are following a downward trend [Durnin 1971].

Protein intakes are well within the RNI recommendations, bordering on being high rather than low. Calcium intakes of the boys in the all the studies do not reach the RNI (11-14 year old) value of 1000mg/d. However, the boys in the Kent, London and Dundee studies were ages 9-12 years and it may be equally appropriate to use the RNI values of the lower age group (7-10 years). If so, the calcium intakes are adequate. The girls in all the studies have calcium intakes that reach the RNI (11-14 year old) value of 800mg/d except those in the London and Dundee studies. Since the girls in the London study were 10-11 years old then they could be assessed with the RNI set for 7-10 years old. This value is 550mg/d indicating an adequate intake.

The children in the Glasgow study have the highest reported iron intakes of 12.5mg/d which is above the RNI set

for boys aged 11-14 years, but below that set for girls. The Kent and London studies who recruited children aged 9-11 years could have their iron intakes compared to the RNI set for the 7-10 year old range which is 8.7mg/d. When this is considered the children in these studies have adequate iron intakes. The children in the Northumberland and Dundee studies report iron intakes that are below the RNI value.

The Vitamin A intakes reported in the Kent and Northumberland studies are similar to the RNI of 600μg retinol equivalent per day. The dietary intakes of Vitamin A in the children recruited to the London and Dundee studies fall below this. If comparisons are made with the RNI value set for 7-10 year olds, 500μg retinol equivalent per day, then the children in the London and Dundee studies have adequate intakes. The only exception are the girls in the London study whose reported intakes are 460μg retinol equivalent per day.

The reported intakes of Vitamin C in all the studies when compared to the RNI are well within the reference point of 35mg/d. The only exception are the girls in the Dundee study who have a reported intake of 28mg/d.

The intakes of thiamin, riboflavin and nicotinic acid reported in all the studies are well above the RNI values recommended.

An indication of dietary quality rather than quantity can be given by calculating the average nutrient intake per 1000 kilocalories energy consumed. Expressing in this way eliminates the effects that body size and physical activity have on dietary intake. Body size and physical activity can affect the absolute intakes of nutrients merely because more

or less is eaten [Cook 1973].

1.1.7 FACTORS AFFECTING DIETARY INTAKES

There are many factors which affect patterns of food intake, for example regional differences of where an individual lives. If an individual lives by the sea, the opportunity of consuming fresh fish is that much higher. Nutrient and dietary intakes can be reflected by regional patterns. The DHSS study [DHSS 1989] of diets of British school children reflected regional differences in consumption of fruit juice. The children in London consumed more than those in Scotland.

Socio-economic status can affect dietary intake [Johnson 1980]. A parent in social class V by definition of work, will earn less money and therefore spend less absolutely on food, but more as a percentage of total income. National studies have shown that social class of parent and height difference of child persists among primary school children [Rona 1978]. The reason for these differences is not known but nutritional intake is suspected to be a part of it.

Dietary intakes are affected by ill-health and disease. In chronic disease metabolic demand for substrate increases. For example, leukaemia is treated with chemotherapy which kills the offending cells. With this treatment other cells are also damaged. The initial chemotherapy treatment results in an increase in metabolic demand and comes from the need for tissue regeneration and in children, the continuation of growth. The requirements for substrate and co-factors will therefore be greater. Once on maintenance chemotherapy however, metabolic demand is seen to decrease [Bond 1991].

1.1.8 SUMMARY

The diet provides the body with the necessary energy and nutrients needed to meet the metabolic demand for existing tissues and for tissue synthesis. Dietary intake can be measured but to be measured accurately the researcher and recorder must use sufficient skill and care. The adequacy of dietary intakes can be assessed using dietary reference points of adequacy.

1.2. GROWTH

The definition and measurement of growth is reviewed. The extent to which nutritional status affects growth is discussed.

1.2.1 DEFINING GROWTH

Growth may be defined as a constellation of changes characterised by changes in form and function, [Jackson and Wootton 1990]. A change in form includes increases in stature, mass and body weight which then also alters body composition. As a human body grows, there is an increase in function, namely the refinement and differentiation of organs and tissues which affect body mass. The increase in function increases the ability to perform more elaborate tasks such as hand/eye co-ordination.

To achieve the increase in form, substrate and co-factors must be available from which the appropriate materials can be drawn to fuel the synthesis. The already existing tissue has in addition, metabolic demands of its own. Thus the more cells multiply or enlarge to increase the existing tissue, the greater the demand for metabolic maintenance.

There are two different types of tissue; regenerating and non-regenerating. With regenerating tissue such as skin and

blood, the cells continually die and are replaced. Non-regenerating tissue is different in that once a cell is formed, it lasts most of the animal/human lifetime. Once the growth period of the cell is over these tissues, eg. muscle and nerve, are unable to manufacture any new cells. Cells whether they be regenerative or non-regenerative, contribute the basic units to an increase in mass. The accumulation of an increase in number or size of cells creates an organ. The organ enables the human body to maintain a wider range of functions of greater complexity.

The increase in cell size or number of cells can be assessed indirectly by measurements of height and weight. These measurements repeated over a period of time will provide the rate at which growth has taken place. The two variables of height and weight describe different dimensions of growth. Growth is said to have ceased when height no longer increases. Height ceases to increase once maturity is reached. However, weight is different. This variable is able to increase and decrease before and after maturity has been reached. The fluctuation of weight is largely due to the relative increase or decrease of physical activity in relation to dietary intake.

Poor growth occurs when conditions within the body or within the external environment are not suitable. When this occurs, potential body size is not reached at maturity. If the conditions become more favourable before maturity, then growth will continue, [Tanner 1978].

The interaction of nutritional intake and metabolic demand on growth is critical. At a cellular level, if substrate is unavailable to meet the metabolic demand for

mitosis or meiosis, then growth will not occur.

Increases in height and weight indicate that in addition to maintenance needs, substrate has been made available for growth. Comparing actual increases in height and weight with quantities recommended will illustrate whether enough substrate has been made available. Nutritional status and growth are therefore very closely linked.

1.2.2 MEASUREMENT OF GROWTH

Growth can be quantified by measurements of height and weight. The measurements of height and weight taken at regular intervals with the same technique will show the pattern of growth and velocity with which it takes place. These individual measurements of the child when plotted on standard growth charts enable the detection of abnormal growth [Tanner 1966].

Standards are used to assess relative rates of growth, which have been developed from longitudinal studies [Tanner and Whitehouse 1976]. The more carefully measurements are taken, the less measuring-error occurs. Measurements of growth taken over shorter intervals rather than longer ones will increase the percentage measurement error of the difference between the readings. If measurements of height are taken at intervals less than 6 months it is difficult to be sure that measured differences are real [Tanner 1976]. The time of year that the measurement is taken will also affect reliability of the measurement. Children grow faster in height in the Spring and Summer compared to Autumn and Winter [Marshall 1971].

Body compositional changes during growth are not reflected in height and weight measurements. More complex

techniques are required to measure body composition. Skin fold measurements are a well established technique used to estimate fat mass, from which fat free mass can be derived.

There are now a variety of other techniques to measure body composition, [Lukaski 1985], but some of these are unable to be used in children or on a regular basis, such as radioactive tracer elements in determining total body water. For children, skinfold measurements are the most suitable and provided the investigator has the appropriate training and experience, there can be precision within 5% [Lukaski 1985].

Derived equations for converting skinfold thickness to fat mass that are suitable for children, should be used [Brook 1971]. Although the reliability of this equation to estimate body composition of children, has not been demonstrated directly, it is the most reliable available at the present time.

1.2.3 GROWTH AND NUTRITIONAL STATUS

The relationship between growth and nutritional status is well established. The measurements of height and weight are used to describe nutritional status.

Weight for age in children as an index is widely used to assess protein energy malnutrition and over-nutrition, especially in infancy when length measurements may be difficult. A major limitation of weight for age being used as an index of nutritional status is that it does not take into consideration height differences. Children with a low weight for age are not necessarily wasted if their weight is less than normal.

Waterlow's classification of malnutrition included both

weight and height, and differentiates between nutritional stunting, when weight may be appropriate for height, and wasting, when weight is low for height [Waterlow 1977].

This index using weight and height is more sensitive to changes in nutritional status than height for age being used alone. A disadvantage of weight for height is that it classifies the child with poor linear growth as 'normal'. Thus it may be preferable to use weight for height and height for age [Waterlow 1977]. If there have been extended periods of inadequate substrate availability, height is the variable that will suffer and so height for age is a useful index of nutritional status in children. As an index it can be used to assess the extent of stunting relative to a child's full growth potential.

When growth is interrupted for a period of time, regaining the deficit in height in full may not be possible. If the reason for growth cessation in a child is poor energy intake, then growth will only continue if extra energy is provided. The extra energy provided must be enough to allow for catch up growth, [Jackson and Wootton 1990]. If the child has a deficiency in growth hormone, then height can only be regained with extra growth hormone being provided.

1.2.4 SUMMARY

The monitoring of height and weight indicates how well or poorly a child is growing. For growth and development to occur the metabolic demand for existing tissues and for tissue synthesis must be met by dietary intake. Growth patterns in children can be measured and assessed using standard growth charts and this in turn will reflect nutritional status.

1.3. SHORT STATURE

The determinants of short stature are reviewed followed by a review of dietary intakes in short children and the consequences of being small. Finally the treatment of short stature is discussed, in particular the treatment with growth hormone and its effect on dietary intake and metabolic demand.

1.3.1 DEFINING SHORT STATURE

Stature is the measurement of leg length, trunk length and standing head height, more commonly known as the height of an individual. Short stature in children can be defined as a child with a height below the 3rd centile when compared to reference standards [Tanner and Whitehouse 1976].

1.3.2 DETERMINANTS OF SHORT STATURE

Height variations within and between populations are well documented [Tanner 1981]. At least 1.3% of the Wessex regions' childhood population are short [Voss 1989]. Many genetic, hormonal, metabolic or environmental factors play a role in its determination.

The genetic determinant of short stature is a result of inherited DNA passed on at conception. These genes develop body size and shape as a result of interaction with the environment. There is more genetic control of shape rather than of stature. This is chiefly because shape describes the distribution of the cells and stature describes the size to which cells and tissues will increase. The size of cells can alter and increase throughout childhood up to and into maturity. Every child will have a different genotype and ideally might require a different environment in which to achieve optimal growth [Waterlow 1985].

Organic disease can interfere with childhood growth patterns. Impaired growth is a recognised complication of many chronic diseases during childhood such as asthma [Cohen 1948, Falliers 1961] and eczema [David 1989, Massarano 1993]. Asthma and eczema are conditions in childhood that may require treatment with corticosteroids. In children, corticosteroids block the multiplication of cartilage cells and cause growth to cease. With other chronic disease such as leukaemia, treatment with radiation especially to the child's head in association with brain tumours, can cause growth hormone deficiency [Leheup 1988]. This will therefore leave the child with short stature unless growth hormone treatment is commenced.

Chromosome and congenital endocrine disorders such as Turners syndrome and growth hormone deficiency result in short stature. With the absence of organic disease short stature still occurs in our population and if not genetic, is as a result of other factors.

Short stature can result as a consequence of inadequate nutrition, [Tanner 1976]. More commonly under-nutrition is perceived to result in a child becoming thin and wasted which of course can happen, but poor growth and short stature are also clinical manifestations of nutritional inadequacy.

In many populations the period at which the child is most at risk of becoming malnourished is 6 months to 3 years [Pelto 1989]. Weaning occurs around 3-6 months with the introduction of solid foods and less milk being given. In the case of breast feeding, very often the mother's lactating ability declines as the prolactin levels of the

body decrease. It is at this same age that infants have a high risk of developing infections and combined with poor food intake associated with the problems of weaning, will develop malnutrition and thus poor stature.

This phenomena is more readily seen in developing countries but the West has the same problems evolving in a different way. There is a high prevalence of inappropriate eating behaviours occurring from the fear of obesity for example or from food restrictions that parents impose on themselves and infants as a consequence of food allergy, whether it be real or perceived [Ferguson 1990]. This is chronic under-nutrition but manifested in a completely different way.

Children having suffered long term nutritional inadequacy will develop short stature and if dietary intervention is left too late, then optimal height potential will not be reached. However, stunted children have demonstrated 'catch-up' growth with nutritional intervention [Prader 1963].

Children from different socio-economic levels differ in stature, [Miller 1972; Smith 1980]. Social class can be defined as social inequality whereby power, wealth and prestige are unequally shared between members of a society. All three variables have an impact on stature. Wealth or family income buys the food that children eat and the house that they live in. Occupational status can be linked to this, since this denotes a certain amount of income. Income and wealth are also linked to power and prestige. The more wealthy an individual is, the more power and prestige they generally have.

It is known that children living in the same environment but having parents from different socio-economic backgrounds, have different statures. Those children in the higher socio-economic group are taller than those in the lower socio-economic group and this variation may be due to nutritional intake [Goldstein 1971].

Various studies have indicated that socio-economic status affects growth and stature of an individual [Rona 1978, Amirhakimi 1974]. Studies have also indicated that social class differences in stature may perhaps be disappearing. A nationwide sample of Swedish urban children followed longitudinally during a 9 year period found no significant differences in height with social status as defined by father's occupation and income [Lindgren 1976]. A study among Norwegian school children in 1975 also showed similar findings [Brundtland 1980].

1.3.3 DIETARY INTAKES OF SHORT STATURE CHILDREN

There is little data regarding the dietary intakes of children and adults with short stature. Only two studies have investigated dietary intakes of short stature children [Okada 1979, Walker 1990]. The study of Okada [Okada 1979] looked at the relationship of energy intake and height increment in normal height children, children with short stature and children with growth hormone deficiency being treated with growth hormone. The study reports the short stature children consuming more energy expressed per kg body weight than the normal height and growth hormone deficient children on treatment.

Data also suggests that a decreased energy intake in growth hormone deficient children receiving treatment can

induce unresponsiveness to the growth hormone treatment. The suggestion from this data is that nutritional intake exerts an influence on the activity of IGF-1, which is an intermediary of growth hormone, necessary for skeletal growth. Growth hormone needs an intermediary in its action on the bones to make them grow and this is known as IGF-1 (for insulin-like growth factor). The IGF-1 is secreted by the cells of the proliferative zone of the cartilage, and growth hormone appears to both increase the number of these cells and also turn on their IGF secretion. Further work has supported this theory. A study examining the relationship between plasma IGF-1 factor concentration, nutrient intake and height in healthy Equadorian male children has demonstrated that height is strongly influenced by nutritional intake [Lopez-Jaramillo 1992]. The plasma levels of IGF-1 were also much lower in children who were shorter.

A question that remains to be answered is; does a short child eat more or less than a taller child. The dietary intakes of short stature children are not known. It is also not known whether the dietary intakes of these children alter when treated with growth hormone and once treatment has finished.

1.3.4 THE CONSEQUENCES OF BEING SMALL

There are many consequences of being small, both physiological and psychological. Evidence suggests that impaired growth and development during fetal life and early infancy will substantially increase the risk of heart disease, stroke and the associated conditions of hypertension and diabetes, [Barker 1992]. That is to say

that being small at birth and upto one year of age relates strongly to mortality from cardiovascular diseases later in adult life. The relationship of retarded growth in fetal life and infancy with cardiovascular disease may be a consequence of an insult occurring at a critical, sensitive period early in life which results in long term changes in physiology or metabolism.

The psychological consequences of being small can be quite marked, [Skuse 1987]. With children, self-image is formed from sensory impressions and ability to do tasks. In establishing this personal identity, other individuals reactions are very important. By the age of 8 years a child has a view of themselves as a person which has developed as a result of individuals attitudes to them and on differences in their own appearance [Harter 1982]. By this age the appearance of short stature begins to be more obvious. The psychological effect resulting from short stature in an otherwise normal and healthy child can often go unrecognised.

A child who is different in appearance to their peers will attract attention. Short children have to cope with an identity which is determined by their size. Adults will also reinforce this identity with short children. A child who is short or small for their age can be treated as if younger by parents, teachers and other adults [Alley 1983]. They are often helped more than a child of normal height which can cause immaturity and insecurity as well as other behaviourial problems.

Children who are short may have a high incidence of learning problems [Gold 1978] and often view themselves as

less popular and happy than their peers. This may lead to social withdrawal, aloofness and isolation [Gordon 1982].

A child of short stature will develop their own personality, based on how they perceive themselves and how others see them. Between the ages of 4 to 15 years of age, physical features are one of the most important determinants of self esteem [Kelnar 1990]. Small or short stature children are more aware of their physical appearance and as a result can develop a sense of being inadequate and incompetent.

They are often excluded from participating in peer group activities on the basis of their size. This occurs more characteristically for boys rather than for girls. Boys are seen to be the protectors and gain dominance, prestige, success and leadership from their physical appearance. Thus being short and small as a boy, reinforces the feeling of inadequacy which is not so marked with a girl.

Short stature children cope with their size in different ways. Some may never cope and continue with feelings of insecurity and incompetence into adulthood. Others will cope by excelling in an independent skill, such as playing a musical instrument, or by befriending a larger child who will protect them. Others assume the role of being the 'clown' amongst their peers, often with an extrovert pattern of behaviour.

1.3.5 TREATMENT OF SHORT STATURE

The population in general has individuals of varying heights. There will be some of those individuals when corrected for age who will be short when compared to others. Organic disease or endocrine disorders can be a

cause of shortness but even when allowance has been made for this there remain many short stature individuals who are disproportionately short.

Growth hormone is necessary for normal growth. Synthesised and stored in the anterior pituitary gland, growth hormone is secreted from birth throughout childhood and into adult life. It has profound effects on skeletal bone growth as well as on body composition and its lipolytic action is well documented [Goodman 1974]. Growth hormone induces lipolysis in adipocytes thus reducing the amount of adipose tissue in the body. The same hormone also stimulates cell division and enlargement of muscle cells thereby increasing the muscle mass and visceral tissue in the body.

Growth hormone thus increases the skeletal and muscle tissue in the body whilst decreasing the fat mass. With the increase in muscle tissue which is metabolically more active than fat tissue, metabolic demand might be expected to rise.

Short stature occurring as a result of a chromosome disorder, for example, Turners syndrome in females and endocrine disorders such as growth hormone deficiency has usually been treated with growth hormone therapy.

Growth hormone is species-specific. From 1958 to 1985, growth hormone had to be extracted at autopsy from human pituitary glands for any individual who needed such treatment. However, in 1985 biosynthetic growth hormone became available through pioneering work using DNA technology, and was granted a licence for clinical use. This has revolutionised its use in the field of endocrinology. The increased availability of growth hormone

has now meant that children who are short without the presence of a defined organic disorder can be considered for growth hormone therapy under appropriately controlled and monitored conditions [Sawisch 1986].

The goal in medicine is health and normal functioning of the individual. Because short but otherwise normal children have no underlying medical pathology, many clinicians feel uncomfortable about growth hormone therapy. However, there are those that feel a child does not need an underlying pathology to suffer because of short stature. These children may not be functioning in a normal psychological way.

There has been a wealth of data investigating how growth hormone is secreted in short but otherwise normal children and the ideal way that growth hormone should be given. Recent studies have been carried out investigating the use of growth hormone with short but otherwise normal children [Hindmarsh 1987; Colwell 1990; Lesage 1991] and how best to provide the therapy [Kastrup 1983], to increase body height.

In the recent work with adults, the basal metabolic rate expressed per kg of lean body mass increases significantly when growth hormone therapy was given to growth hormone deficient adults [Solomon 1989, Sonksen 1991]. Much of the data available on the measurement of basal metabolic rate is with adults and children who are having growth hormone therapy as a result of growth hormone deficiency. There is only limited data on how basal metabolic rate alters when short but otherwise normal children are given growth hormone therapy, [Walker 1990, Gregory 1991]. With therapy these short children increase in height, accumulate more muscle

and lose body fat but what occurs after treatment has been completed is unknown. Growth hormone is used as an option for treatment of short stature and has recently been suggested as a possible treatment for short stature in developing countries [Nutrition Reviews 1992].

1.3.6 SUMMARY

Short stature can be defined as a child with a height of <3rd centile or less than -2.0 SD for height. Genes, hormones, environment and dietary intake all play a role in controlling short stature which once developed can have tremendous psychological impact on the individual. There is only one paper which has looked at the dietary intakes of short stature children. No work has been carried out investigating the dietary intakes of children who are short and are being treated with growth hormone.

1.4. SUMMARY OF THE REVIEW OF LITERATURE

There is literature which has investigated dietary food patterns and nutrient intakes in children of normal height. The studies that have been carried out indicate that meat followed by confectionery, chips, cakes and biscuits are the major contributing foods to energy in the diet. The literature also suggests that the children are consuming adequate nutrient intakes of protein, thiamin, riboflavin and nicotinic acid when compared to standard references.

Growth will only occur if the nutritional supply required for the synthesis of new tissues and for already existing tissues is met. The literature reviewed identifies that not only is the quantity of a nutrient in the diet important in providing this but also the quality or types of foods consumed in the diet. If these requirements are not met then short stature can result. The review gives evidence that little is known about food patterns and nutrient intakes of short stature children.

In summary, the literature reviewed exposes the need to investigate fully the dietary food patterns and nutrient intakes of short stature children. It is also unknown if food pattern, nutrient intake and metabolic demand for energy alter when short stature is treated with growth hormone.

1.5. AIMS OF RESEARCH

* Does the energy intake of short stature children originate from different food sources compared to normal height children?

* Are there differences in energy and nutrient intake between short stature and normal height children that may be a cause of limited growth?

* Do short stature children consume enough dietary energy to satisfy the metabolic demand required for tissue maintenance and growth?

* What are the metabolic demands for energy in short stature children?

* Growth hormone has profound lipolytic and anabolic effects on the body. To what extent do food patterns, nutrient intake and metabolic demand in short children change in response to growth hormone treatment? Is dietary intake adequate to fulfil the requirements of metabolic demand and rapid growth?

CHAPTER 2

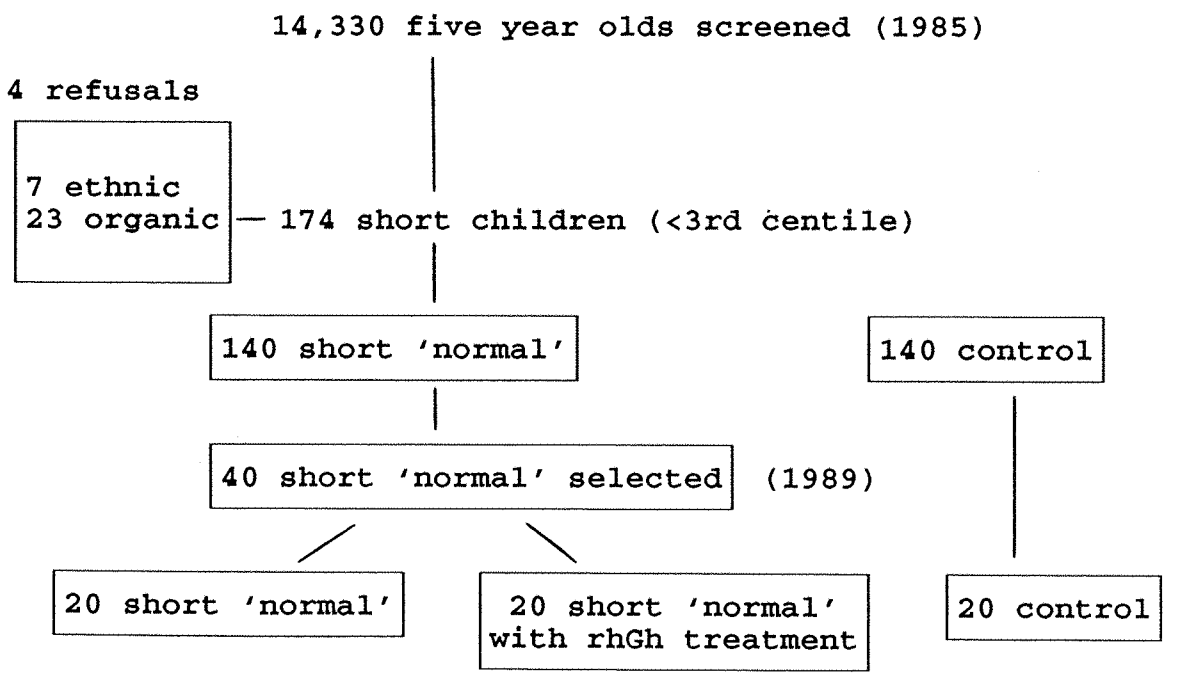
2. METHODS

This chapter is divided into five sections. The first section describes the children who took part in the study. The second section provides the details of how the anthropometry and body composition were measured. Thirdly, the Basal Metabolic Rate measurement method is described. The fourth section gives details of how the dietary assessments were made and the fifth and last section describes the statistical analysis.

2.1 SUBJECTS

All the children in this research were part of an ongoing study called the Wessex Growth Study [Voss 1989]. The Wessex Growth Study started in 1985 and screened approximately 14,000 5 year old children in the Southampton area for shortness, to identify and monitor physical growth, and psychological development.

Figure 2.1 THE WESSEX GROWTH STUDY



From the 14,000 screened, 140 of these children were on or below the 3rd centile for height when plotted on the Tanner and Whitehouse growth charts. These short children were then matched to normal height controls who were of the same sex and age and were also attending the same school (see figure 2.2 over which illustrates two children are matched for sex and age). Then in 1989, from the main study, a small cohort of 40 short children were taken. With parental and ethical consent, 20 of these short children were randomly allocated to either treatment with growth hormone (rhGh), 30 Iu/m^2/week. ('Genotropin', Kabi-Pharmacia, Milton Keynes, U.K.).

In the first year of treatment, the effect of rhGh on BMR and body composition was investigated and compared to measurements made in the short untreated children (Walker 1990).

During the second year of treatment the work described in this thesis started investigating metabolic demand and the nutritional intakes in these children. At this time some children had dropped out from the Wessex Growth Study, either having moved out of the area or because they were no longer attending hospital appointments.

In total, 17 children in their second year of growth hormone treatment and 17 of their normal controls were selected for the study. All the children were aged 9-10 years. Any child who became unwell during the study period or was unable to maintain reliable records of BMR or dietary intake was excluded from the study (accounted for four children). In each group there were finally 7 girls and 8 boys except in the normal height group where there were

Figure 2.2 Height difference of two boys matched for age

7 girls and 9 boys. All the children were caucasian. Each parent of the child was contacted by letter or telephone which outlined the nature of the study. Verbal consent by all the parents was given. Each child and one or both parents were then seen for an interview at home or, at the hospital, if they were attending an appointment in connection with the Wessex Growth Study.

Ethical approval for this study was granted by the Southampton Hospitals and South West Hampshire Health Authority Ethical Committee.

2.2 ANTHROPOMETRY AND BODY COMPOSITION

Height was measured using a Holtain stadiometer. Weight was determined in light clothing using digital electronic scales. Both the stadiometer and electronic scales were regularly calibrated against a standard. The stadiometer measured to within 1mm and the electronic scales to within 100g. Where the stadiometer and electronic scales were unavailable, a wall mounted height rule was used and a portable balance scale. These other pieces of equipment were calibrated using a standard metre rule and known standard weights. Standard deviation scores were determined for height [Tanner and Whitehouse 1983].

Skinfolds were measured at four sites on each child, biceps, triceps, subscapular and supra-iliac. The measurements were made by a single observer using Holtain callipers and a steel tape measure [Edwards 1955]. Before the children in the study were measured, training of the author was undertaken using adult and child volunteers.

Body density was determined using the equations of Brook (1-11 years) [Brook 1971] and percentage body fat was

estimated using the equations devised by Siri [Siri 1956]. Fat free mass values were then determined using body weight.

In this study, the error in measuring skinfolds was considered. Each child had three measurements taken at each skinfold site and the midway point of the three measurements recorded. The same method of measurement was consistently used for each child.

2.3 BASAL METABOLIC RATE

Basal Metabolic Rate (BMR) was determined in all the children using indirect calorimetry. The system was an open system with a ventilated hood. BMR was defined as by Dubois [Dubois 1936] as the minimum energy expenditure required for maintaining essential bodily functions, under standardised resting conditions following an overnight fast in a neutral thermal environment. All measurements were performed between 0730 - 1030 hours in a research room at the hospital. Prior to the children attending for the measurement they were asked to fast overnight and not to have breakfast before arriving in the morning. They were able to drink water if they wanted. The early morning tests enabled the children to be at school by 0900, thus not causing any school work to be missed.

During the measurement, the subjects were asked to be as relaxed and still as possible. This was helped by listening with a Walkman to selected music or story tapes.

Thermo-neutral conditions were achieved by maintaining the room at 22-24°C and adjusting with a fan heater as necessary. The measurements were conducted for a minimum period of 30 minutes, after a stable energy expenditure had been achieved.

Before starting the measurement the Datex Deltatrac Metabolic Monitor was calibrated for gas and pressure.

Allowance was made for the Deltatrac to warm up prior to calibration for at least 30 minutes. Room pressure was noted using the wall barometer in the research room. The gas used for the calibration contained 95% oxygen and 5% carbon dioxide. This procedure took approximately 4-5 minutes to perform and was repeated before each measurement was taken.

The Deltatrac was connected to a printer which typed out results every minute. The printout recorded and printed out the inspired and expired gases, flow rate, respiratory quotient (RQ) and energy BMR every minute. The mean BMR was calculated from the 30 measurements recorded over the 30 minutes.

The transparent perspex hood was laid over the head of the child. Between the hood and pump was a disposable filter. The filter was regularly changed and the hood cleaned with Hibi spray after each measurement.

Predicted BMR values were calculated for age, gender, weight and height using the equations of Schofield [Schofield 1985] and compared to the actual measures of BMR.

2.4 DIETARY ASSESSMENT

A weighed dietary intake was recorded for a period of 7 days in the manner described by Marr [Marr 1971].

To record dietary intake, digital electronic kitchen scales weighing up to 1000g with a tare facility were used. The scales were made by Soehnle, were battery operated (1.5v x 6), and measured in 1g divisions up to 64g and then in 2g divisions up to 1000g.

Each child used a food diary to record their daily intake. The diary was a very small plastic covered file containing typed instructions on how to use the diary, and loose leaf pages ruled to record the time of a meal, type of food or drink taken, weight of food or drink, a column to describe the food still further and a food code column.

Each child was seen with one or both parents for 30 minutes and instructed on how to use the scales and how to complete the diary. Emphasis was placed on keeping as much a normal food pattern as possible for the 7 days. Where the child stayed for school meals, the school catering staff, like the parents, were given a duplicate set of scales and record diary and instructed on how to use them. Discussions were also held to check the contents of unusual foods that the children may have had in the school meal. Figure 2.3 illustrates the school meal system where the weighing of the food took place once the child had chosen the meal.

During the dietary assessment week, parents were contacted by telephone to identify any problems. At the end of the assessment period the diaries were collected and checked with the child and parent for accuracy and clarity.

The food and drinks were coded according to the McCance and Widdowson tables [Paul and Southgate 1978] with any other data needed being obtained from manufacturers information. Using a computerised database (Microdiet) analysis was carried out to determine the estimated metabolisable energy and nutrient intake over the 7 day period.

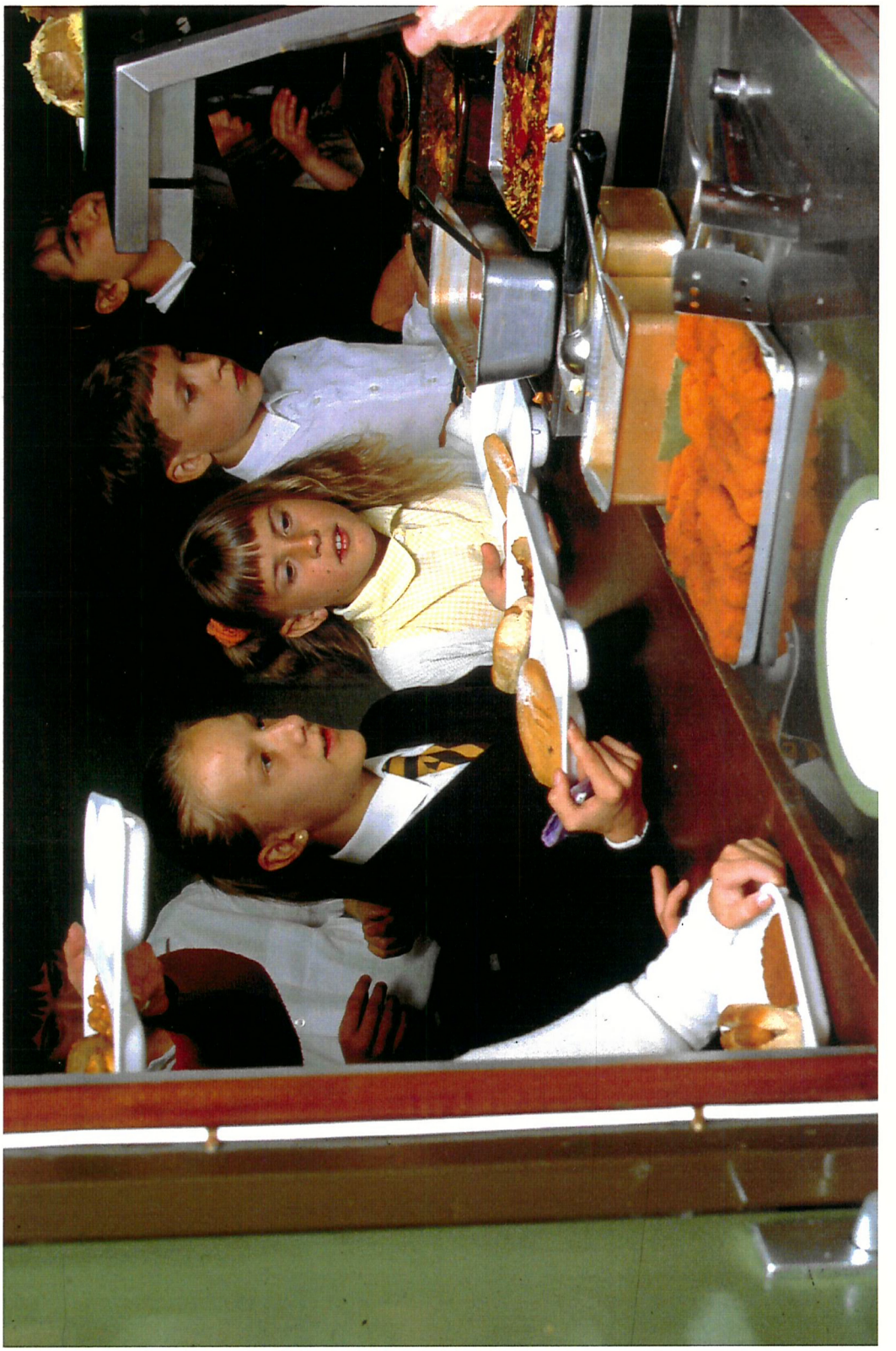


Figure 2.3 School children choosing their midday meal

Patterns of food intake were also identified with each child. The food and drink (coded according to the McCance and Widdowson tables) were assigned to major food group categories used within these tables [Paul 1978]. The total energy from each food group was determined manually.

In the past there has been some concern expressed by Livingstone [Livingstone 1990], about the validity of the collection of dietary data. For randomly selected populations, there may be a tendency to under-report in some instances. The reliability of dietary records depends very much on the motivation of both the recorder and the researcher. This group of children were supported well by their parents who appeared to be extremely keen to help with the study. Great care was taken at all times to minimise any of the recognisable errors associated with the assessment method.

Bingham has reviewed many of the methodologies associated with the measurement of dietary intake and while recognising the difficulties involved, identifies that with sufficient skill and care, reliable results can be obtained by using a 7 day weighed inventory and recommends its use under appropriate circumstances [Bingham 1987]. Ideally for validation, dietary intake should be repeated and where possible compared with other methods. Within this study this was not possible and may be of less importance when comparing group means.

2.5 STATISTICAL ANALYSIS OF DATA

All the data collected was collated on floppy disks, using the computer program SPSS/PC data entry II.

Statistical analysis was performed using the SPSS/PC

statistics version 4.1 program. The mean, standard deviation and range for all the variables were derived. The Tukey test was applied to test for statistical significance between the three groups of children [Cohen 1979].

There are inherent difficulties associated with the statistical analysis of differences between groups, where variables are expressed relative to a point of reference (i.e. EAR, RNI) or relative to a second variable (i.e. body size). In particular the problem of comparing the differences between variables expressed in percentage terms (i.e. percentage energy from carbohydrate) is recognised by the author.

In this study, alternative approaches to the statistical analysis have been extensively explored, including non-parametric analysis, transformation of the data, and co-variant analysis. However, none of these approaches yielded different interpretations of the data from that gained by simple parametric comparisons.

Therefore, the statistical analysis of the differences between groups in this thesis are presented in a uniform way. Assuming that the data is normally distributed and that where relative expressions of variables are presented, the relationship between the two component variables is constant and linear over the observed range. Where no statistical difference was found, care was taken to discuss trends.

2.6 METHOD OF INVESTIGATION FOR EXPERIMENTAL WORK

In this thesis three strategies were used to assess dietary intake.

* The first strategy assessed which foods provide the energy in these three groups of children.

* The second strategy assessed energy and other nutrient intake compared with the Dietary Reference Values and relative to body size.

* The third strategy assessed energy intake in relation to metabolic demand.

CHAPTER 3

3. FOOD PATTERNS IN THE SHORT STATURE CHILDREN, SHORT STATURE CHILDREN RECEIVING GROWTH HORMONE TREATMENT AND NORMAL HEIGHT CHILDREN.

3.1 INTRODUCTION

The quality of diet taken in early childhood has been of great concern. A child's health and optimal development depends on dietary intake [Boulton 1981]. Concerns have centred on the dietary habits of school children, since there is more freedom today to choose where and what to eat. The Department of Health and Social Security commissioned a dietary survey of British school children in 1983 to monitor the effect of the Education Act in 1980 [DHSS 1989]. The Education Act in 1980 enabled individual schools to take charge and be responsible for the provision of meals. The survey was to elucidate whether the Act was affecting dietary intake and food patterns to the extent that health, stature and possible learning capacity in later life were being affected.

For there to be concern, the degree of inadequacy of the diet does not have to be so great as to cause signs of frank deficiency. Lesser degrees of inadequacy may have effects on a child's ability to resist infection and their learning capability. These may relate to the childs overall ability to grow. Dietary food patterns and quality of diet can provide information on the adequacy of the diet and an indication of the potential for children's growth and development [Allen 1991].

The DHSS in 1989, thus reported on the dietary intakes

and food patterns of 3591 children aged between 10-14 years in England, Wales and Scotland. This study provides a reference to which other studies can be compared.

Food choice and behaviour is acquired in many ways, most of which develops as a result of exposure to these foods in the home with parents, grandparents and siblings. Food choice also develops as a result of social interaction with peers at school.

There have been several studies investigating food patterns and nutrient intakes of normal height children [DHSS 1989, Hackett 1984, Adamson 1992] but not in short children. It is therefore known what the normal population of children choose to eat but not what the population of short children choose to eat. Do this group of children choose a pattern of foods which is sufficiently different that it may ultimately be affecting their growth?

In any dietary survey it is important to report nutrient intakes but equally important is the reporting of intakes of foods or food patterns. People choose foods to eat and not nutrients.

For dietary advice to be effective for any group of the population, a knowledge of their present eating habits must be known and dietary advice given in terms of these foods. Since the dietary food patterns of short children are not known, the challenge presented to the clinician or dietician becomes more difficult, when faced with advising on the diet of a short child.

This study aims to determine the dietary habits and food patterns of short stature children, short children receiving rhGh treatment and normal height children.

3.2 METHODS

3.2.1 SUBJECTS

Fifteen short stature children who were <-2.0 SDS, 15 short stature children originally <-2.0 SDS but with rhGh treatment, were now -1.5 SDS and 16 normal height children with a SDS of +0.24 were included in this study. These children were part of a larger study ongoing in Wessex, the detail of which is outlined in Chapter 2. The participation was voluntary.

3.2.2 DATA COLLECTION

Standard procedures were followed to measure each child's dietary intake for 7 days. The detail of the procedure is outlined in the Methodology section. For the week of the recording, each child was issued with electronic digital scales and a specially designed notebook. Where necessary, children were visited at home and at school. The food diaries were coded using a computerised data base of Microdiet.

As the aim of the study was to compare the contribution of selected food groups to energy, all the foods were grouped. The food groups used largely follow the groupings given in 'The Composition of Foods' [Paul and Southgate 1978]. The 'meat' group included any pastry that was consumed as part of a meat item, for example meat pies. The milk group included milk, yoghurt and cheese. The 'cakes and biscuits' group also included puddings such as ice-cream and rice pudding. The 'confectionery' group included chocolate, chocolate bars, sweets, sugar, jams, still and carbonated drinks, fruit squashes, iced lollies and milk shakes. The 'crisps and chips' included those foods only,

all other potato was included in the 'vegetable and fruit' group.

The percentage energy contribution of foods to the total energy intake was calculated in the three groups of children. A further analysis was carried out of the energy contribution from food groups per kg body weight to determine whether body size contributed to derived differences in energy contributed by each food group.

3.2.3 STATISTICAL ANALYSIS

Values are presented as means with standard deviation and range. All the data collected was analysed using SPSS software. The Tukey test was used to test for statistical significance.

3.3 RESULTS

The subject characteristics are given in Table 3.1. There were no significant differences in age between the groups. The untreated short children were significantly shorter than both the short children who had been treated with rhGh for up to 18 months (6%, $p<0.05$) and the control group (14%, $p<0.01$). The short untreated children were also lighter than both other groups.

Table 3.2 illustrates the food patterns seen in the short stature children, short treated children and normal height children. Meat and meat products contributed more energy to the diet of normal height children than to either groups of short children. Both groups of short stature children consumed 8.5% energy from meat, 21% lower compared to the normal height children. The percentage energy contribution made by milk and milk products was lowest in the short children being treated with rhGh at 7.8% with the short

untreated children consuming only 0.4% more than this. The normal height children had 10.8% of their energy provided by milk. There was no significant difference between the groups in the percentage energy provided by meat and milk.

The percentage energy contributed by cakes and biscuits was 16.7% in the short treated group, 23-30% higher compared to the normal height and short untreated group. When comparing the percentage energy provided by confectionery, both groups of short children have more energy provided by these foods, 20.8% ($p<0.05$) in the short treated group and 19.6% in the short untreated group, when compared to the normal height children at 13.1%. The difference between the short children and the normal children did not attain statistical significance. The short treated children have a 37% higher percentage energy contribution from confectionery compared to normal height children.

The percentage energy contribution of chips and crisps to the total energy was 12-13% in both groups of short children, half as much again as the normal height children who had a percentage energy contribution of 7.9% ($p<0.01$) from crisps and chips.

The percentage energy contribution provided by cereals and bread to the total energy of the diet was 15.7% in the short stature group and 16.3% in the short treated group. This was 16% lower than the normal height group who had an 18.9% energy contribution from cereals and bread.

Vegetables and fruit had a much lower percentage energy contribution in the short untreated group and short treated group 4.9% ($p<0.05$) and 5.6% ($p<0.01$) when compared to the normal height group who had half as much energy again being

contributed by vegetables and fruit. The normal height children had a 50% increase in vegetables and fruit contributing to energy.

When the percentage energy contribution of food groups was expressed in terms of body size, the pattern is different (see Table 3.3). Meat and meat products contributing to energy and expressed per kg body weight per day were all comparable in all three groups. The same pattern was seen with milk and milk products.

However, when the food group of cakes and biscuits were analysed, the short treated children consumed 55 kJ/kg/d ($p<0.05$), 40% more than the normal height children and short untreated children who were consuming 33 and 35 kJ/kg/d respectively. A difference was also seen with the confectionery group, except both groups of short children tended to consume more than the normal height children. The short treated group consumed 71 kJ/kg/d ($p<0.01$) which was 52% higher in confectionery consumption compared to the normal height children. The short untreated group consumed 60 kJ/kg/d ($p<0.05$), 40% lower in confectionery consumption compared to the normal height group.

Again both groups of short children consumed more crisps and chips when expressed relative to body size. The short treated group consumed 50% ($p<0.05$) more crisps and chips compared to the normal height children. The short untreated group consumed 40% more when compared to the normal height group, but this difference did not attain statistical significance.

Cereal and bread consumption when expressed in terms of body size were very similar in all three groups, 52 kJ/kg/d

in the short group, 55 kJ/kg/d in the short treated group and 49 kJ/kg/d in the normal height group. The short groups tended to consume more when body size was considered, although statistical significance was not achieved.

Both the short treated and short untreated children consumed less vegetables and fruit than the normal height group when expressed per kg body weight. The short untreated children consumed 34% less vegetables and fruit compared to the normal height children and the short treated children consume 13% less than the normal height children. When expressed per kg body weight and not as percentage energy contribution there was no statistical significance.

Total sugars consumption was much higher in the two short groups of children 5.79g/kg/d ($p<0.05$) in the short treated children and 4.44g/kg/d in the short untreated children, 40% and 20% higher compared to the normal height group. The starch consumption followed a similar pattern when expressed as g/kg/d. The short treated group consumed 19% more than the normal height group and the short untreated children 12% more than the normal height group.

Table 3.1 Subject characteristics of short stature children (SHORT), children of short stature receiving rhGh treatment (SHORT+T) and control children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
	SHORT (1)	**SHORT + T(2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Age (years)	9.17 ± 0.53 8.28 - 10.04	9.39 ± 0.64 8.20 - 10.38	9.66 ± 0.58 8.63 - 10.52	NS	NS	NS
Height (cms)	116.8 ± 2.9 113.4 - 118.0	123.7 ± 4.6 116.9 - 130.8	136.5 ± 4.8 127.0 - 145.0	p<0.05	p<0.01	p<0.01
Height (SDS)	-2.45 ± 0.39 -3.15 - -1.81	-1.51 ± 0.34 -2.22 - -1.02	0.24 ± 0.7 -1.32 - 1.42	p<0.01	p<0.01	p<0.01
Weight (kg)	21.41 ± 3.21 17.20 - 26.30	24.35 ± 2.99 19.50 - 29.80	31.33 ± 6.42 32.70 - 50.00	NS	p<0.01	p<0.01

Table 3.2 Percentage energy contribution of foods to total energy intake of short stature children (SHORT), short children receiving rhGh treatment (SHORT + T) and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
	SHORT (1)	**SHORT + T(2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Meat and meat products (% energy)	8.5 ± 5.1 1.7 - 19.1	8.50 ± 3.8 2.7 - 17.4	10.8 ± 6.3 0.2 - 22.6	NS	NS	NS
Milk and milk products (% energy)	8.4 ± 3.7 1.7 - 14.4	7.8 ± 4.8 0.3 - 25.5	10.8 ± 4.6 2.9 - 20.5	NS	NS	NS
Cakes and biscuits (% energy)	11.6 ± 7.5 7.8 - 37.5	16.7 ± 7.6 7.8 - 37.5	12.7 ± 6.8 0.1 - 24.6	NS	NS	NS
Confectionery, sugar, jam and soft drinks (% energy)	19.6 ± 8.4 5.5 - 32.8	20.8 ± 5.1 10 - 32.7	13.1 ± 9.0 1.8 - 38.9	NS	$p<0.05$	NS
Crisps and chips (% energy)	13.2 ± 4.9 6.3 - 24.4	12.3 ± 5.8 6.2 - 24.4	7.9 ± 5.9 0.1 - 20.6	NS	$p<0.01$	$p<0.01$
Cereals and bread (% energy)	15.7 ± 6.3 3.6 - 26.2	16.3 ± 3.9 9.2 - 24.7	18.9 ± 9.2 2 - 42.7	NS	NS	NS
Vegetables and fruit (% energy)	4.9 ± 2.9 0.1 - 9	5.6 ± 3.3 1.2 - 14	9.9 ± 5.1 1.6 - 19.8	NS	$p<0.05$	$p<0.01$

Table 3.3 Contribution of food groups to energy per kg body weight for short stature children, short children receiving rhGh treatment and normal height children. Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
	SHORT (1)	**SHORT + T (2)**	**NORMAL (3)**	**1 v 2**	**1 v 3**	**2 v 3**
Meat and meat products kJ/kg/d	25 ± 14 7 - 64	29 ± 16 7 - 62	26 ± 18 1 - 52	NS	NS	NS
Milk and milk products kJ/kg/d	24 ± 10 7-36	26 ± 15 1 - 69	26 ± 15 5 - 66	NS	NS	NS
Cakes and biscuits kJ/kg/d	35 ± 26 0.1 - 93	55 ± 25 24 - 107	33 ± 21 0.3 - 82	NS	NS	$p < 0.05$
Confectionery, sugar, jam and soft drinks kJ/kg/d	60 ± 29 20 - 94	71 ± 19 26 - 96	34 ± 24 6 - 98	NS	$p < 0.05$	$p < 0.01$
Crisps and chips kJ/kg/d	38 ± 15 17 - 71	42 ± 21 20 - 85	21 ± 15 3 - 66	NS	NS	$p < 0.05$
Cereals and bread kJ/kg/d	52 ± 18 25 - 82	55 ± 15 32 - 89	49 ± 31 6 - 138	NS	NS	NS
Vegetables and fruit kJ/kg/d	15 ± 10 0.5 - 3.5	20 ± 12 4 - 51	23 ± 16 3 - 56	NS	NS	NS
Sugars g/kg/d	4.44 ± 1.6 1.4 - 6.8	5.79 ± 1.6 2.1 - 8.9	3.53 ± 1.4 1.1 - 6.0	NS	NS	$p < 0.01$
Starch g/kg/d	4.22 ± 0.64 3.2 - 5.7	4.58 ± 0.99 3.3 - 7.5	3.68 ± 1.49 1.3 - 8.1	NS	NS	NS

3.4 DISCUSSION

The purpose of the present study was to examine whether children of short stature consumed a different pattern of foods to normal stature children and whether accelerated growth following treatment with rhGh in children of short stature was associated with any changes in food pattern.

Essentially the results of the present study suggest that the short stature children consume more confectionery, sugar, crisps and chips than the children of normal stature, either when expressed as a percentage energy contribution to total energy or, as the total energy provided by the food, corrected for body size. The findings of this study also show that treatment with rhGh results in a further increase in the energy contributed by foods such as cakes, biscuits, sugar, jam, chocolate, crisps and chips (expressed as a percentage of total energy or amount of energy corrected for body size). Short children already consume an increased percentage energy from these foods compared to that of normal height children, which is increased still further with rhGh treatment. Thus, the food pattern of the short children is sustained but the amount of energy contributed by cakes, biscuits, crisps and chips is increased.

The studies carried out by the DHSS and Adamson [DHSS 1989, Adamson 1992], report the percentage energy contribution by cakes and biscuits to be 8-11% (see Table 1.2). This is similar to the short untreated children at 11.6% and normal height children at 12.7%. The children on rhGh treatment have an increased energy contribution made by cakes and biscuits at 16.7%.

Other studies [Durnin 1971, Hackett 1984] have reported

the percentage energy contribution from confectionery to be 14% but more recently Adamson [Adamson 1992] reported an increase of energy contribution from confectionery to 17% (see Table 1.2). In this study the percentage energy contribution by confectionery observed in both groups of short children was increased at 20% whilst the normal height children had a contribution nearer 14%.

The percentage energy contribution of chips and crisps to total energy intake was recorded by some studies to be 12% [Adamson 1992, Hackett 1984] but others [DHSS 1989] record a contribution nearer 8% (see Table 1.2). Both groups of short stature children in this study had increased intakes of chips and crisps at 12-13% but the normal height children had an intake that compared with the DHSS study of 7.9%.

The contribution of energy provided by meat in this study was similar to the data of other studies (see Table 1.2). Both groups of short children had only half the percentage energy, 8.5%, contributed by meat compared to 16% in other studies [Adamson 1992]. Milk contribution followed a similar pattern. The normal height children had similar amounts of energy provided by milk compared to other studies [Durnin 1974], but both groups of short children had less energy contributed from milk.

The results of this study also indicate that short children whether having rhGh treatment or not consume less vegetables and fruit compared to the normal height children. This trend is seen either expressed as a percentage contribution to energy or as the amount of energy per unit body weight. This cannot be compared to any other studies since no one has recorded vegetable and fruit consumption in

children as a percentage contribution to energy.

Short stature children appear to have a preference for sweet foods in comparison to normal stature children. This idea is strengthened by the short children on rhGh having an increased demand for energy (see Chapter 5.3) and choosing to provide for this by consuming sweet foods.

Food preference may be innate or may be learned. There is evidence that it can be modified during childhood. Birch et al showed that when foods are presented in a positive context, whether sweet or not sweet, food preference can be modified by learning [Birch 1981].

Parents of the normal height children may be more aware of the healthy eating messages and offer more non-sweet foods in a positive context. If so, it is likely that these parents will be using less sugar and less fat in the meals for themselves. Children of this age, (10-11 years) still rely on their adult carers for provision of food and so will be given the same. It may be that the normal stature children have a less developed sweet preference as a result of this modified learning.

The question to be asked is whether this is as a result of an innate preference for sweet food or is it as a result of the environment, with parents and peers only providing these foods in the home, thus reinforcing a preference for them.

The results of this study indicate that the food patterns of short children and short children receiving rhGh treatment are not the same as the food patterns observed in normal height children.

Could the increased quantity of energy provided by

confectionery, sugar, chips and crisps be creating a diet of low nutrient density in short stature children which may then affect growth? One might expect a diet that has an increased quantity of energy provided by sugary and fatty foods to have a lower nutrient density. The following experimental chapter investigates whether this is so.

CHAPTER 4

4. THE NUTRIENT INTAKES OF SHORT STATURE CHILDREN, SHORT CHILDREN RECEIVING GROWTH HORMONE AND NORMAL HEIGHT CHILDREN.

4.1 INTRODUCTION

Children who receive sufficient energy and nutrients to meet their dietary requirements should grow adequately. If requirements are not met then growth will not take place. Nutritional requirements are provided for by substrate and co-factors. Each of these particular substrate and co-factors have specific functions at the molecular level where they provide the material to satisfy the needs of the metabolic demand.

In the past, interest has focused on protein and more recently on the adequacy of dietary energy. Other nutrients such as zinc and iron are recognised as critical elements necessary as components for growth. When looking at nutrient intakes in relation to growth, it is important to realise that it may not just be one component which directly or indirectly affects growth. Nutrients interact with each other to produce a favourable environment within which growth can occur.

If the intake of the specific dietary nutrient(s) meet the requirements suggested, one cannot assume that this is the amount that is absorbed. Other components in the diet may be interacting and inhibit the full absorption of the nutrient in question.

This study aims to determine the adequacy of the nutrient intakes of short children, short children receiving rhGh

treatment, and normal height children in comparison with each other and with recommendations made for these same aged children.

4.2 METHODS

4.2.1 SUBJECTS

The 15 short stature children, the 15 short stature children receiving rhGh, and the 16 normal height children who took part in this study were part of a large growth study unique to Wessex as described in detail in Chapter 2.1. The participation was voluntary.

4.2.2 DATA COLLECTION

Standard procedures were followed to measure each child's dietary intake for seven days. Their intake was recorded using digital electronic scales explained in detail in the Methodology section 2.4. All the children and parents were contacted regularly throughout the week of study. On completion of the dietary record the children and parents were visited at home to collect the scales and food diary and also to ensure there were no ambiguities regarding the food intake.

4.2.3 STATISTICAL ANALYSIS

Values are presented as means with standard deviation and range. All the data collected was analysed using SPSS software.

4.3 RESULTS

The subject characteristics are given in Table 3.1 (refer to chapter 3, page 56). There were no significant differences in age between the groups. The untreated short children were significantly shorter than both the short children who had been treated with rhGh for up to 18 months

(6%, $p<0.05$) and the control group (14%, $p<0.01$). The short untreated children were also lighter than both other groups.

Table 4.1 (page 72) illustrates that the energy intake of the short treated children was 464 kJ/day, a 6% higher compared to the normal height children and 1955 kJ, 24% higher compared to the short untreated children. Energy intakes were compared with the EAR [DH 1991] for each individual. The short treated children and normal height children consumed 105% and 98% of the EAR for energy respectively. The short children reached only 80% of the EAR. The standard reference value of EAR is set separately for boys and girls. Since the study groups were of mixed sex, an average between the two EARs set for boys and girls was taken. The protein intake of the normal height children and short treated children measured in g/day is higher than the short stature children 28% ($p<0.01$), 20% ($p<0.05$) respectively (see Table 4.1). When expressed as a percentage of RNI, the same difference emerges. All the children achieved the RNI set for their age group. The percentage energy taken as protein was similar in both groups of short children, 10.6%. The normal height children took 12.4% of their energy as protein, significantly more.

Fat intakes measured in absolute amounts of g/day were higher in the short treated children compared to the short stature children (21%, $p<0.05$) and normal height children (16%). When expressed as a percentage of energy intake, little difference was observed between the groups. Carbohydrate intake measured in absolute amounts of g/day, was higher in the normal height children but when expressed as a percentage of energy intake, the mean values across all

the groups are very similar.

Tables 4.2 and 4.3 (pages 73 and 74) illustrate the trend of micro-nutrient intakes across the groups. For this study the nutrient intakes were calculated from a 7 day weighed food record and so caution had to be exercised in their interpretation at being representative of the overall nutrient intakes. For micro-nutrient intake assessment, it is recommended that a 4 day weighed food record taken each month over a period of several months provides more accuracy for determining absolute intakes. Nevertheless, the information collected has a value for comparative purposes.

Sodium intake measured in absolute amounts is shown to be lower in the short stature children who had a 27% ($p<0.05$) lower intake than the normal height children. The short treated group had an intake 5% (NS) lower than the normal height children (see Table 4.2). When compared to the Recommended Nutrient Intake (RNI) [DH 1991] set for this age group, the sodium intake in all three groups reach more than the RNI recommended. However, when the sodium intake is expressed per MJ of energy consumed in Table 4.4 (page 75), the differences are not as obvious. The short stature children and short stature children with rhGh treatment have values slightly below the normal height group, 306 mg/MJ and 297 mg/MJ compared to 331 mg/MJ, the differences do not achieve conventional statistical significance.

Potassium intake, when expressed in absolute amounts of mg/day, reveal the short stature children had a lower mean intake compared to the normal height children (24%, $p<0.01$) and short treated children (22%, $p<0.05$). The short treated and normal height children achieved RNI values of 120% and

123% respectively, whereas the short stature children reached a value of 93%. When compared and expressed per MJ energy intake, then values between the groups were very similar, as shown in Table 4.4.

Calcium intakes when expressed in absolute amounts were lower in the short stature children compared to the normal height children (37%, $p<0.01$), and short treated children (20%, NS). The short treated and normal height children achieved RNI values of 114% and 145%, whereas the short stature children achieved 91%. Expressing calcium intake as mg/MJ, the short stature children had a lower mean value compared to the normal height children (21%, $p<0.05$). The short treated children had a lower mean value compared to the normal height group (24%, $p<0.01$).

The short children consumed less magnesium in absolute amounts compared to the short treated children (25%, $p<0.01$) and normal height children (26%, $p<0.01$) (see Table 4.3). The short stature children achieved 80% of the RNI whilst the short treated group and normal height group achieved values of 107% ($p<0.01$), and 109% ($p<0.01$) respectively. When expressed as mg/MJ, the values of each group were very similar.

When comparing intakes of iron in absolute amounts, the short treated children consumed a lower mean intake of 6.81 mg/day, compared to the short treated group and (27%, $p<0.01$) and normal height group (22%, $p<0.05$). The short stature group only reached 78% of the RNI whereas the short treated children and normal height children reached 154% and 100% RNI respectively. When expressed as mg/MJ consumed, the mean values of iron intake were very similar across the

groups, 1.1 mg/MJ respectively.

Copper intakes seen from Table 4.3 indicated that the short stature children had a lower mean intake of 0.91 µg/day compared to the short treated children (26%, $p<0.01$) and normal height children (24%, $p<0.05$). All three groups reached the percentage RNI for 9-10 year old children. Expressing the intakes as µg/MJ consumed, gave very similar mean values throughout the three groups of 0.15 µg/MJ.

The zinc intakes of the three groups of children illustrated that when expressed in absolute amounts of mg/day, the short children had a lower intake of 4.57 mg/day compared to the short treated children (25%, $p<0.05$) and normal height children (34%, $p<0.01$). All three groups of children had percentage RNI just below 100%. The normal height group consume 99% RNI, short treated children 87% of RNI and the short stature children 98% RNI. When expressed as mg/MJ energy consumed, the short treated children consumed a lower mean intake of zinc compared to the normal height children (22%, $p<0.01$). The short untreated children also consumed a lower intake of zinc in mg/MJ compared to the normal height children (18%, $p<0.01$).

Tables 4.5 and 4.6 (pages 76 and 77) illustrate the vitamin intakes and trends of the three groups of children. When expressed in absolute amounts, Vitamin A intake had a lower mean value in the short children compared to the normal height and short treated children although the difference was not statistically significant (see Table 4.5). Table 4.7 (page 78) illustrates that when Vitamin A intake was expressed as µg retinol eq/MJ, the short group had a lower mean value compared to the short treated group

(26%) and normal height group (21%). The differences however are not statistically significant. Both the normal height children and short treated children reach over 100% RNI while the short stature children consume only 83% of the RNI.

Vitamin D intakes expressed in absolute amounts of µg/day showed that the short children had a lower mean value compared to the short treated children (20%) and normal height children (15%). These differences did not attain statistical significance. When expressed as µg/MJ in Table 4.7, all the three groups had very similar mean values of 0.19 µg/MJ. There is no Vitamin D RNI set for individuals who are normal and active, only for those who are housebound. Comparisons with these standards therefore cannot be made.

When comparing Vitamin E intakes the short stature group had a lower mean value in mg/day, compared to the short treated group (19%) and the normal height group (8%). When the intakes were expressed per MJ energy consumed, the short group consume the highest intake, 5% more than the short treated children and 15% more than the normal height children (see Table 4.7). The differences between these values were not statistically significant. There are no dietary reference standards for Vitamin E intakes as this is related to the polyunsaturated fatty acid intake which may vary widely. Ranges of acceptable intakes are only available.

Vitamin B_1 intakes when expressed in absolute amounts were lower in the short children, compared to the normal height children (39%) and short treated children (22%). All

three groups of children attain the RNI for age. When expressed as intake per MJ consumed, all three groups had similar values. No significant differences existed between the groups.

The levels of Vitamin B_2 intakes when expressed as mg/day followed the same trend as Vitamin B_1. The short stature children had a lower mean value compared to the normal height group (40%, $p<0.05$) and short treated group (21%, NS). All the groups met the RNI for age. When dietary intake of Vitamin B_2 was expressed per MJ energy consumed, the short stature children and short treated children had very similar values. The normal height children had an higher value (37%) compared to both short stature groups, but the difference was not statistically significant.

Nicotinic acid intakes, expressed in absolute amounts, mg Eq/MJ, showed that the short stature children had a lower mean value compared to the normal height children (32%) and short treated children (24%). All mean intake values met the RNI. When intake was expressed as mg/MJ, all mean values were similar. No statistically significant differences were observed between the three groups.

The daily intake of Vitamin C, mg/day, was lower in the short children, compared to the short treated children (52%, $p<0.01$) and normal height children (19.5%, NS). When the Vitamin C intakes were expressed in mg/MJ the intakes were very similar, the normal height children and short untreated children having intakes of 10-11 mg/MJ and the short untreated children 16.8 mg/MJ.

Vitamin B_6 intake, expressed as µg/day, was lower in the short stature children compared to both the normal height

and short treated children (21%). When intake was expressed as µg/MJ consumed, the short and normal height children had similar mean values with short treated children having higher values. No statistically significant differences were observed between the groups. The RNI for Vitamin B_6 is based on the amount of protein taken in the diet (15 µg/g protein) [DH 1991]. Using the protein intakes observed in the three groups of children, all achieved the RNI set for 10-11 year old children.

Table 4.6 shows Vitamin B_{12} intakes that when expressed in absolute amounts the short stature children had lower mean values compared to the short treated children (37%) and normal height children (33%). The intakes of Vitamin B_{12} reached more than 100% of the RNI in all groups. When Vitamin B_{12} intake was expressed per MJ energy consumed, both the short treated children and normal height children had the similar intakes of 0.32 and 0.34 µg/MJ compared to the intake observed in the short group, 0.25 µg/MJ. These differences were not statistically significant.

Folic acid intakes, Table 4.6, were lower in the short stature group compared to the normal height group (31%, $p<0.05$) and short treated group (17%, NS). When expressed per MJ energy intake, all the three groups had values which were very similar.

Table 4.1 The energy, protein, fat and carbohydrate intakes of short stature children (SHORT), children of short stature receiving growth hormone (SHORT + T), and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
VARIABLES	**SHORT (1)**	**SHORT + T (2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Energy kJ/day (E)	6228 ± 1030 4687 - 9039	8183 ± 1241 6804 - 10826	7719 ± 1218 5842 - 9491	$p < 0.01$	NS	$p < 0.01$
Energy as % of EAR	80 ± 14.4 56 - 109	105 ± 18.8 84 - 148	98 ± 12.9 70 - 117	$p < 0.01$	$p < 0.01$	NS
Protein g/day	40.3 ± 11.4 28.1 - 70.0	50.2 ± 6.6 42.0 - 63.8	56.6 ± 10.9 39.9 - 74.7	$p < 0.05$	NS	$p < 0.01$
Protein as % of RNI	142 ± 40.2 99 - 247	177 ± 23.5 137 - 225	199 ± 38.8 140 - 265	$p < 0.05$	NS	$p < 0.01$
Protein as % of E	10.6 ± 1.5 9 - 14	10.7 ± 1.3 9 - 14	12.4 ± 1.5 10 - 15	NS	$p < 0.01$	$p < 0.05$
Fat g/day	58.9 ± 9.6 45.6 - 80.5	74.5 ± 22 47.9 - 136.3	70.7 ± 12.5 53.2 - 92.9	$p < 0.05$	NS	NS
Fat as % of E	36 ± 4.4 30 - 43	34 ± 5.7 25 - 47	35 ± 5.0 21 - 43	NS	NS	NS
Carbohydrate g/day	210 ± 40.7 135 - 308	285 ± 45.2 213 - 365	258 ± 59.9 182 - 401	NS	NS	NS
Carbohydrate as % of E	53 ± 5.1 44 - 60	55 ± 6.0 44 - 65	53 ± 5.5 44 - 66	NS	NS	NS

Table 4.2 The sodium, potassium, and calcium intakes of short stature children (SHORT), of short stature children receiving rhGh treatment (SHORT + T), and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
VARIABLES	**SHORT (1)**	**SHORT + T (2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Sodium mg/day	1889 ± 533 1150 - 3137	2415 ± 805 1553 - 4717	2559 ± 671 1644 - 4313	NS	NS	$p < 0.05$
Sodium as % RNI	156 ± 44.4 95 - 261	201 ± 67.1 129 - 393	213 ± 59.9 137 - 359	NS	NS	$p < 0.01$
Potassium mg/day	1864 ± 513 984 - 3195	2412 ± 413 1607 - 3216	2464 ± 555 1810 - 3621	$p < 0.05$	NS	$p < 0.01$
Potassium as % RNI	93 ± 25.4 49 - 159	120 ± 20.5 80 - 160	123 ± 28 90 - 181	$p < 0.05$	NS	$p < 0.01$
Calcium mg/day	505 ± 140 355 - 807	634 ± 142.6 317 - 874	802 ± 293 448 - 1542	NS	NS	$p < 0.01$
Calcium as % RNI	91 ± 25.5 64 - 146	114 ± 25.9 57 - 158	145 ± 53.3 81 - 280	NS	NS	$p < 0.01$

Table 4.3 The magnesium, iron, copper and zinc intakes of short stature children (SHORT), of short stature children receiving rhGh treatment (SHORT + T), and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
VARIABLES	**SHORT (1)**	**SHORT + T (2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Magnesium mg/day	160 ± 37 90 - 252	215 ± 45 145 - 323	219 ± 46.0 154 - 317	$p < 0.01$	NS	$p < 0.01$
Magnesium as % RNI	80 ± 18.7 45 - 126	107 ± 22.4 72 - 161	109 ± 22.9 76 - 158	$p < 0.01$	NS	$p < 0.01$
Iron mg/day	6.81 ± 1.83 3.90 - 11.2	9.4 ± 2.44 5.90 - 15.6	8.72 ± 1.54 6.0 - 11.0	$p < 0.01$	NS	$p < 0.05$
Iron as % RNI	78 ± 20.8 45 - 128	154 ± 173.5 67 - 774	100 ± 17.5 69 - 125	NS	NS	NS
Copper µg/day	0.91 ± 0.25 0.50 - 1.60	1.24 ± 0.40 0.78 - 2.40	1.17 ± 0.20 0.89 - 1.60	$p < 0.01$	NS	$p < 0.05$
Copper as % RNI	129 ± 36.2 67 - 228	177 ± 57.3 111 - 342	158 ± 49.6 1.7 - 228	$p < 0.05$	NS	NS
Zinc mg/day	4.57 ± 1.37 3.10 - 8.60	6.11 ± 1.47 4.22 - 9.36	6.96 ± 1.25 4.80 - 9.00	$p < 0.05$	NS	$p < 0.01$
Zinc as % RNI	98 ± 124.6 44 - 543	87 ± 20.8 60 - 133	99 ± 18 68 - 129	NS	NS	NS

Table 4.4 The sodium, potassium, calcium, magnesium, iron, copper and zinc intakes per MJ energy consumed, of short stature children (SHORT), of short stature children receiving rhGh treatment (SHORT + T), and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
	SHORT (1)	**SHORT + T(2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Energy kJ/d	6228 ± 1029 4687 - 9039	8133 ± 1242 6808 - 10826	7719 ± 1218 5842 - 9491	$p < 0.01$	NS	$p < 0.01$
Sodium mg/MJ	306 ± 83 175 - 503	297 ± 113 203 - 693	331 ± 71 262 - 459	NS	NS	NS
Potassium mg/MJ	294 ± 36 209 - 353	295 ± 39 223 - 358	322 ± 68 199 - 442	NS	NS	NS
Calcium mg/MJ	81 ± 20 53 - 126	78 ± 20 45 - 118	103 ± 30 54 - 159	NS	$p < 0.01$	$p < 0.05$
Magnesium mg/MJ	25 ± 3 19 - 29	26 ± 5 20 - 36	28 ± 4 22 - 35	NS	NS	NS
Iron mg/MJ	1.0 ± 0.16 0.8 - 1.4	1.1 ± 0.43 0.8 - 2.3	1.1 ± 0.16 0.8 - 1.3	NS	NS	NS
Copper µg/MJ	0.14 ± 0.02 0.10 - 0.17	0.15 ± 0.03 0.10 - 0.23	0.15 ± 0.02 0.11 - 0.19	NS	NS	NS
Zinc mg/MJ	0.73 ± 0.15 0.56 - 1.20	0.70 ± 0.15 0.47 - 1.04	0.90 ± 0.14 0.60 - 1.13	NS	$p < 0.01$	$p < 0.01$

Table 4.5 The Vitamin A, D, E, B_1 and B_2 intakes of short stature children (SHORT), of short stature children receiving rhGh treatment (SHORT + T), and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
VARIABLES	**SHORT (1)**	**SHORT + T (2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Vitamin A µg retinol eq/day	416 ± 290 123 – 1011	771 ± 652 227 – 2634	665 ± 325 227 – 1606	NS	NS	NS
Vitamin A as % RNI	83 ± 57.9 24 – 202	154 ± 130.3 45 – 526	133 ± 65.3 45 – 321	NS	NS	NS
Vitamin D µg/day	1.27 ± 0.88 0.25 – 3.30	1.60 ± 0.98 0.41 – 3.90	1.49 ± 0.69 0.56 – 2.80	NS	NS	NS
Vitamin D as % RNI	–	–	–	–	–	–
Vitamin E mg/day	3.90 ± 1.06 2.60 – 6.70	4.84 ± 1.35 1.87 – 7.42	4.24 ± 1.06 2.20 – 7.10	NS	NS	NS
Vitamin E as % RNI	–	–	–	–	–	–
Vitamin B_1 mg/day	0.91 ± 0.23 0.52 – 1.27	1.17 ± 0.34 0.67 – 1.83	1.49 ± 1.01 0.56 – 4.98	NS	NS	$p < 0.05$
Vitamin B_1 as % RNI	206 ± 76.0 3.1 – 295	291 ± 85.9 167 – 457	283 ± 131 3.2 – 545	NS	NS	NS
Vitamin B_2 mg/day	1.03 ± 0.29 0.70 – 1.60	1.37 ± 0.41 0.62 – 1.84	1.73 ± 1.07 0.92 – 5.49	NS	NS	$p < 0.05$
Vitamin B_2 as % RNI	102 ± 28.7 70 – 161	136 ± 41.1 62 – 184	173 ± 106.7 92 – 549	NS	NS	$p < 0.05$

Table 4.6 The nicotinic acid, Vitamin C, B_6, B_{12} and folic acid intakes of short stature children (SHORT), of short stature children receiving rhGh treatment (SHORT + T), and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
VARIABLES	**SHORT (1)**	**SHORT + T (2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Nicotinic acid mg Eq/MJ/day	11.24 ± 1.95 7.4 - 14.0	14.85 ± 3.68 10.1 - 23.0	16.46 ± 10.23 5.9 - 52	NS	NS	NS
Nicotinic acid as % RNI	169 ± 29 112 - 207	223 ± 55.3 153 - 348	249 ± 154.7 90 - 787	NS	NS	NS
Vitamin C mg/day	69.7 ± 55.8 15.0 - 191.0	148 ± 139 23 - 543	86.6 ± 57.5 20.7 - 190.0	$p < 0.01$	NS	NS
Vitamin C as % RNI	232 ± 186 50 - 636	356 ± 276.9 76 - 856	305 ± 183.6 69 - 633	NS	NS	NS
Vitamin B_6 mg/day	0.80 ± 0.26 0.45 - 1.55	1.02 ± 0.22 0.51 - 1.44	1.02 ± 0.29 0.65 - 1.74	NS	NS	NS
Vitamin B_6 as % RNI	132 ± 26.3 45 - 155	159 ± 31.1 12 - 144	141 ± 29.2 65 - 174	NS	NS	NS
Vitamin B_{12} µg/day	1.68 ± 0.83 0.6 - 3.4	2.68 ± 2.21 1.1 - 9.6	2.53 ± 1.17 1.2 - 5.5	NS	NS	NS
Vitamin B_{12} as % RNI	168 ± 82.9 60 - 340	194 ± 123.4 26 - 543	253 ± 117.2 122 - 548	NS	NS	NS
Folic acid µg/day	94.7 ± 37.7 44.0 - 179.0	115.4 ± 22.8 93 - 188	138.2 ± 52.2 82 - 286	NS	NS	$p < 0.05$
Folic acid as % RNI	63 ± 25.1 29 - 119	76 ± 15.4 62 - 125	92 ± 34.8 54 - 190	NS	NS	$p < 0.05$

Table 4.7 The vitamin intakes per MJ energy consumed of short stature children (SHORT), of short stature children receiving rhGh treatment (SHORT + T), and normal height children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
	SHORT (1)	**SHORT + T(2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Energy kJ/d	6228 ± 1029 4687 - 9039	8133 ± 1242 6808 - 10826	7719 ± 1242 5842 - 9491	$p < 0.01$	NS	$p < 0.01$
Vitamin A µg retinol eq/MJ	65.6 ± 43 21 - 156	89.2 ± 62.2 31.6 - 257	87.0 ± 42 33 - 203	NS	NS	NS
Vitamin D µg/MJ	0.20 ± 0.13 0.04 - 0.54	0.18 ± 0.10 0.05 - 0.40	0.19 ± 0.09 0.07 - 0.37	NS	NS	NS
Vitamin E mg/MJ	0.63 ± 0.13 0.40 - 0.84	0.58 ± 0.15 0.26 - 0.97	0.55 ± 0.14 0.24 - 0.86	NS	NS	NS
Vitamin B_1 mg/MJ	0.14 ± 0.04 0.07 - 0.27	0.14 ± 0.04 0.08 - 0.21	0.18 ± 0.10 0.09 - 0.51	NS	NS	NS
Vitamin B_2 mg/MJ	0.16 ± 0.05 0.07 - 0.26	0.16 ± 0.05 0.07 - 0.26	0.28 ± 0.28 0.11 - 1.27	NS	NS	NS
Nicotinic Acid mg/MJ	1.83 ± 0.40 1.3 - 2.9	1.81 ± 0.39 1.2 - 2.5	2.0 ± 0.98 1.0 - 5.3	NS	NS	NS
Vitamin C mg/MJ	10.1 ± 8.8 2.3 - 32	16.8 ± 16.4 0.34 - 61	11.5 ± 8.2 2.5 - 32	NS	NS	NS
Vitamin B_6 µg/MJ	0.12 ± 0.02 0.09 - 0.18	0.18 ± 0.21 0.07 - 0.97	0.12 ± 0.03 0.08 - 0.21	NS	NS	NS
Vitamin B_{12} µg/MJ	0.25 ± 0.12 0.08 - 0.50	0.32 ± 0.23 0.11 - 0.93	0.34 ± 0.18 0.17 - 0.79	NS	NS	NS
Folic Acid µg/MJ	14.1 ± 3.1 9.0 - 20.0	14.1 ± 1.8 11.0 - 18.0	17.8 ± 6.2 9.0 - 35.0	NS	NS	NS

4.4 DISCUSSION

The nutrient intakes of the short stature children, short stature children on rhGh treatment and the normal height children were compared with existing Dietary Reference Values (DRV) [DH 1991] of nutrients. The values used for comparison were the Reference Nutrient Intakes (RNI). This level was decided on since this level of intake of nutrient is almost enough for every individual, even those who have high needs.

For most children the RNI is considerably higher than needed. If children are consuming the RNI of a nutrient they are unlikely to be deficient in that nutrient. If a child is habitually consuming less than the LRNI they will almost certainly be deficient. In this cross-sectional study seven children in the short stature group had individual intakes of vitamin A and zinc that were below the LRNI.

Protein intakes observed in these three groups of children are very similar and in fact are comparable to intakes of other British schoolchildren [DHSS 1989]. The short stature children have lower protein intakes than the normal height children but are still consuming more than the RNI. In the past, recommended intakes have been based on the assumption that people in the U.K. who are accustomed to taking at least 10% of their energy as protein, are not protein deficient. The RNI is based on data from nitrogen balance studies and has a value that is much lower than the recommendations set in 1989 [DHSS 1989].

Fat, carbohydrate and protein are the main substrates used to provide energy for metabolic demand. The

recommendations of the COMA report [DHSS 1984] and DRV report [DH 1991] suggests that total fat intake should not exceed 35% of energy. The children in the present study consumed 34-35% of energy from fat, 12% less than the British schoolchildren recruited to the DHSS survey.

Carbohydrate intake of the children in the present study was higher than that reported for British schoolchildren (see Table 1.4). The energy for metabolic demand in these three groups of children is being provided mainly by carbohydrate.

Intakes of protein and carbohydrate despite being lower in the short stature groups, when compared in absolute amounts, are similar when expressed as a percentage of energy intake. The protein intakes when measured as a percentage of energy intake, are significantly different between the short groups of children compared to the normal height children.

Calcium, magnesium and zinc; when expressed as the amount per MJ energy consumed, give mean values in both the short stature children and short stature children receiving growth hormone that are lower than the normal height children. Zinc intakes in both groups of short children when expressed as the amount per MJ of energy consumed, are significantly lower. These two minerals and trace element are responsible for growth and development in the human body [Gibson 1990]. Calcium and magnesium are stored primarily in the bones and are minerals needed to build bone mass [Gibson 1990]. Other properties involve membrane transport in energy metabolism [Gibson 1990]. Zinc is known to be a nutrient needed specifically for growth and development [Cousins 1986].

Assumptions that there is a direct link between these nutrients and level of growth obviously cannot be made but these observations have been made by Gibson [Gibson 1989] and Walravens [Walravens 1983] regarding zinc intake.

Sodium and potassium intakes are also determined by total food intake, hence energy intake. Both short stature groups then have similar intakes when expressed as mg/kJ, which are not significantly different to the normal height children.

Other studies have reported dietary intakes of nutrients (see Table 1.4). These dietary surveys have reported calcium within a range of 767 mg/day to 966 mg/day which is similar to the values observed in the normal height children in this study. The short stature groups have values well below this. These same studies (see Table 1.4) report intakes of iron in children to be in a range of 8.6 mg/day to 11 mg/day. The short children fall below this at 6.8 mg/day, but the other two groups have similar intakes to these studies.

Vitamin A intakes have been reported (see Table 1.4). The values measured in the short treated children and normal height children compare well to these studies, 771 and 665 µg retinol eq/day compared to the highest reported value of 997 µg [Cook 1973] and lowest reported value of 460 µg [DHSS 1989]. The short untreated group have an intake 10% below the lowest reported value.

The Vitamin B_2 intake of children has been reported by other studies (see Table 1.4). It is only the short untreated children that have an intake in mg/day that is observed to be lower than the values reported in other studies. These same studies have recorded Vitamin C intakes from 28 mg/day to 49 mg/day which are much lower than the

intakes observed in this study.

This study suggests that the nutrient intakes, as measured in absolute amounts, are different between the three groups of children. The most significant differences of absolute nutrient intake were seen between the short untreated and normal height children. Very little difference exists between the short treated and normal height children. In absolute amounts energy intake was decreased in the short children compared to the normal height children. Because of this one would expect all other nutrients in the diet to also be lower when expressing in absolute quantities. Any difference in nutrient intake is reflected by the consumption of more or less food. Despite the decreased nutrient intakes in the short children, as measured in absolute amounts, all nutrients attained 63% or more of their RNI.

When comparing the diets of the children qualitatively, as mg or g of nutrient per MJ energy intake, there is no difference between any of the groups. Thus the nutrient density of the food consumed by the short children, short treated children and normal height children are the same.

The previous chapter concluded that short and normal height children have different food patterns. The short children and short children receiving rhGh treatment consumed an increased quantity of confectionery, crisps and chips, in comparison to the normal height children. So, here we have an anomaly arising - the short children consume more confectionery and fatty foods compared to the normal height children but the nutrient density of the diet remains the same.

There can be two debates: the first is that both these conclusions are correct. Certain foods that the children are consuming are masked by other foods. For example, the nutrient density of such foods as confectionery are the same as the base diet. This would therefore mean that no matter how much confectionery is consumed in the diet, the overall nutrient density will remain the same. Only if the increased energy intake were provided by sugar, would there be any noticeable effect on the nutrient density of the diet. The second debate is that one of the assumptions is incorrect. The two assumptions are that the original food was recorded accurately and that the nutrients derived and estimated are also accurate. It is more likely that the weight of the foods consumed are accurate since these can be measured. Nutrient intakes however, can only be estimated and predicted from the weight of the food and use of food tables. Errors are more likely to be introduced here.

The study illustrates that expressing nutrient intakes in various ways gives rise to different interpretations of the quality of the diet. The short children and short treated children have diets of similar nutrient density despite consuming more of the confectionery and fatty foods.

CHAPTER 5

5. ENERGY INTAKE AND BASAL METABOLIC RATES IN CHILDREN OF SHORT STATURE AND THOSE RECEIVING GROWTH HORMONE

5.1 INTRODUCTION

In earlier work, Basal Metabolic Rate (BMR) has been measured in short but otherwise normal children before and 6 months after treatment with growth hormone and the results compared with children of normal height [Walker 1990]. It was found that before any treatment with rhGh, the BMR in the normal short children was significantly greater than in taller children when expressed relative to body mass (i.e., kJ/kg body mass/day). The interpretation of the data suggested that relative to size, the energy demands of short children were greater than those of taller children.

Observations with children in Jamaica found that these short children consume more energy for their size than children of normal height [Walker 1990].

The findings from these two studies raise important questions concerning food intake and poor growth - are short children short because their requirements for energy are relatively increased or short as an adaptation to a limited availability of food? It has been generally assumed that shortness is a consequence of a reduced food intake. These findings suggest that this assumption needs to be questioned critically.

Anthropometric indices are often used to define nutritional state [Soares 1991]. However, the work by Soares indicates that these measurements alone cannot characterise nutritional state. The measure of metabolic

function has to be taken into consideration.

It is also unclear the extent to which any changes in metabolic rate observed in short stature children might be an inherent characteristic or may just reflect their immediate past nutritional intake.

This chapter discusses measurements of energy intake and BMR taken at the same time in three groups of children - short normal, short normal being treated with rhGh, and children of normal stature.

5.2 METHODS

5.2.1 SUBJECTS

The 15 short stature children, the 15 short stature children receiving rhGh, and the 16 normal height children who took part in this study were part of a larger growth study unique to Wessex as described in detail in Chapter 2.1. The participation was voluntary. Further explanation on the subjects and amount of growth hormone that was being given is in Chapter 2.1.

5.2.2 DATA COLLECTION

Dietary assessment and Basal Metabolic Rate were measured at the same time in each child. Basal Metabolic Rate was determined in all the children using indirect calorimetry following the procedure detailed in the Methodology Chapter 2.3. Each child had their height, weight and skinfolds measured in order to determine predicted metabolic rate, percentage body fat and lean body mass. More detail on these measurements are given in Chapter 2.2.

Standard procedures were followed to measure each child's dietary intake for seven days. The dietary intake was recorded using digital electronic scales, explained in

detail in the Methodology Chapter 2.4.

5.2.3 STATISTICAL ANALYSIS

Values are presented as means with standard deviation and range. All the data collected was analysed using SPSS software. The Tukey test was applied to test for statistical significance.

5.3 RESULTS

The subject characteristics are given in Table 5.1. There were no significant differences in age between the groups. The untreated short normal children were significantly shorter than both the short normal children who had been treated with rhGh for up to 18 months (6%, $p<0.05$) and the control group (14%, $p<0.01$). The short normal children were lighter than the control group, both in terms of body mass (32%, $p<0.01$) and they tended to be leaner as indicated by lower percent body fat (20%, $p<0.05$). The short treated children also exhibited lower body weights (22%, $p<0.01$), fat free mass (13%, $P<0.01$) and percent body fat (39%, $p<0.01$) than the control group. The treated children had greater lean body mass (20%, $p<0.01$) and also tended to be heavier (14%, $p<0.05$) and leaner (18% lower percent body fat, $p<0.05$) than the untreated children of short stature.

There were significant differences in BMR between the groups (Table 5.2) with the short untreated children expending about 1000kJ/day less than both other groups when expressed in absolute terms. When BMR was expressed in relation to body weight, both the short stature groups exhibited greater BMR values than the control group (untreated 14%, $p<0.05$; treated 20%, $p<0.01$). Even when

corrected for differences in lean body mass, the highest values were observed in the short stature groups (Figure 5.1). The BMR of the treated short stature group expressed per unit of body weight or LBM was not significantly different from that of the untreated short stature group. Although the control group and untreated short stature group had a BMR similar to that predicted for age, gender, body weight and height; the BMR of the short stature group receiving treatment was 17% greater than predicted.

The short treated children consumed about 500kJ/day more than the control children and about 2000kJ/day more than the short untreated children when expressed in absolute terms (Table 5.2). Expressed per unit of body weight, the short treated children consumed more than the short untreated children (14%, $p<0.05$) and control children (32%, $p<0.01$, Figure 5.2). When the energy intake was expressed as a ratio to the BMR, the highest values were observed in the short treated children although the differences between the groups did not attain statistical significance (Table 5.2).

Table 5.1 Subject characteristics of short stature children (SHORT), children of short stature receiving rhGh treatment (SHORT + T) and control children (NORMAL). Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
VARIABLES	**SHORT (1)**	**SHORT + T (2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
Age (years)	9.17 ± 0.53 8.28 - 10.04	9.39 ± 0.64 8.20 - 10.38	9.66 ± 0.58 8.63 - 10.52	NS	NS	NS
Height (cms)	116.8 ± 2.9 113.4 - 118.0	123.7 ± 4.6 116.9 - 130.8	136.5 ± 4.8 127.0 - 145.0	$p < 0.05$	$p < 0.01$	$p < 0.01$
Height SDS	-2.45 ± 0.39 -3.15 - -1.81	-1.51 ± 0.34 -2.22 - -1.02	0.24 ± 0.7 -1.32 - 1.42	$p < 0.01$	$p < 0.01$	$p < 0.01$
Weight (kg)	21.41 ± 3.21 17.20 - 26.30	24.35 ± 2.99 19.50 - 29.80	31.33 ± 6.42 23.70 - 50.00	NS	$p < 0.01$	$p < 0.01$
% Body fat	16.4 ± 4.4 8.3 - 22.7	12.3 ± 3.3 7.0 - 17.8	20.34 ± 6.29 12.6 - 35.2	NS	$p < 0.01$	NS
Lean body Mass (kg)	17.77 ± 2.01 14.50 - 21.40	21.29 ± 2.51 17.60 - 26.30	24.56 ± 2.91 22.00 - 32.00	$p < 0.01$	$p < 0.01$	$p < 0.01$

Table 5.2 Energy Intake and Basal Metabolic Rate of short stature children (SHORT), children of short stature receiving rhGh treatment (SHORT + T) and control children (NORMAL) Values are expressed as means with s.d. and the range underneath.

				STATISTICAL SIGNIFICANCE		
Energy Intake	**SHORT (1)**	**SHORT + T(2)**	**NORMAL (3)**	**1 v 2**	**2 v 3**	**1 v 3**
kJ/day	6228 ± 1029 4687 - 9039	8183 ± 1242 6808 - 10826	7719 ± 1218 5842 - 9491	$p < 0.01$	NS	$p < 0.01$
kJ/kg body wt/day	295 ± 53 200 - 346	339 ± 51 238 - 420	257 ± 65 117 - 348	NS	$p < 0.01$	NS
kJ/kg LBM/day	353 ± 59 255 - 466	327 ± 60 279 - 501	319 ± 64 182 - 414	NS	$P < 0.01$	NS
Basal Metabolic rate						
kJ/day	4077 ± 384 3480 - 4500	4932 ± 533 4456 - 5460	5192 ± 6.27 4120 - 6810	$p < 0.01$	NS	$p < 0.01$
kJ/kg body wt./day	194 ± 30 136 - 247	204 ± 15 181 - 229	170 ± 26 125 - 200	NS	$p < 0.01$	$p < 0.05$
kJ/kg LBM/day	232 ± 29 177 - 268	233 ± 19 196 - 270	212 ± 23 169 - 247	NS	$p < 0.05$	NS
As % predicted	103 ± 11 85.4 - 123.6	117 ± 7 106 - 125	107 ± 9 86 - 125	$p < 0.01$	$p < 0.01$	NS
Energy Intake : BMR	1.54 ± 0.31 1.15 - 2.07	1.66 ± 0.25 1.30 - 2.07	1.50 ± 0.24 1.02 - 1.83	NS	NS	NS

Figure 5.1 Basal Metabolic Rate of children of short stature (SHORT), children of short stature receiving rhGh treatment (SHORT + T) and control children (NORMAL) expressed per unit lean body mass. Individual values are shown with bar indicating mean value for group. SHORT + T significantly different from NORMAL $p < 0.05$.

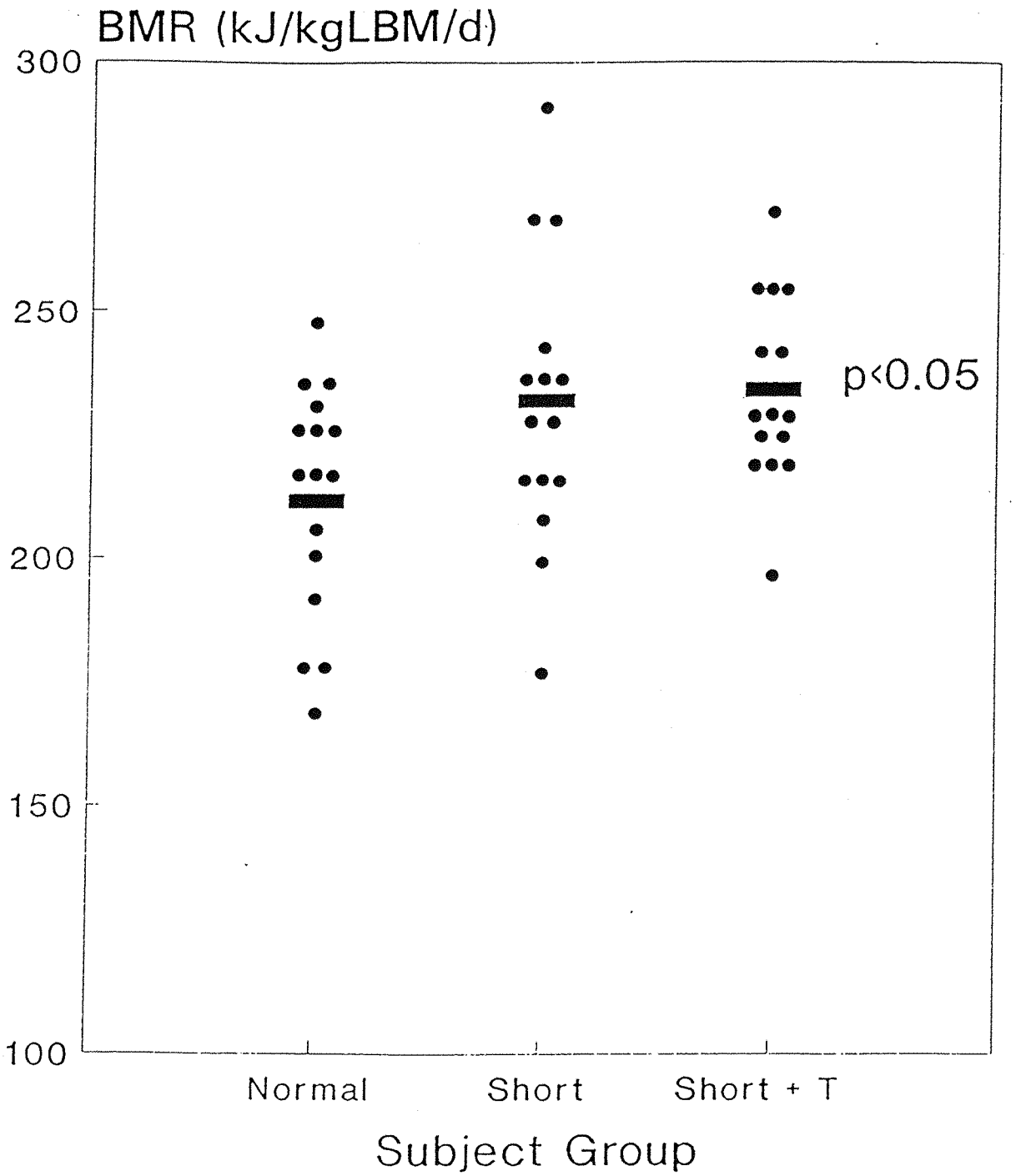
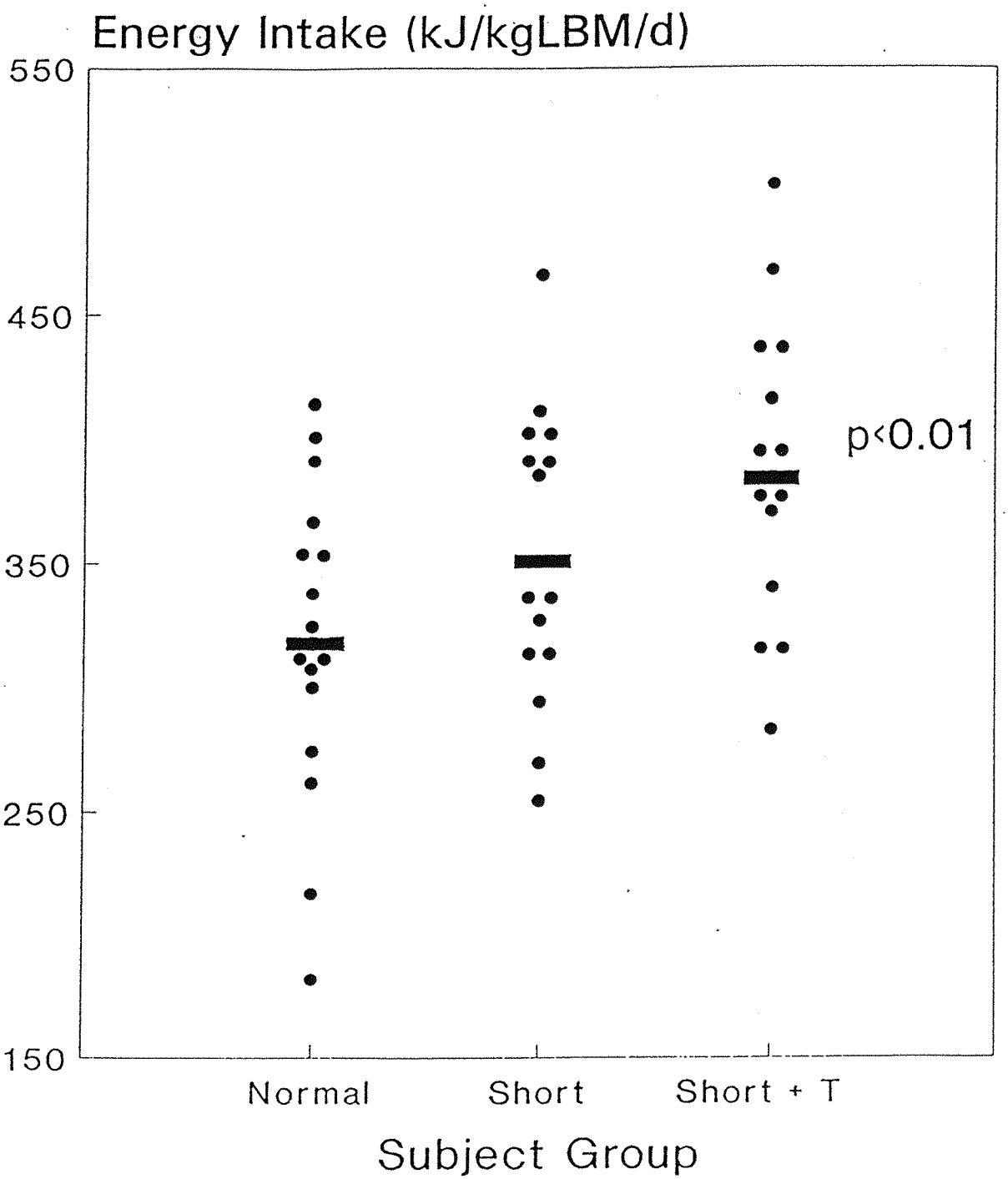


Figure 5.2 Energy intake of children of short stature (SHORT), children of short stature receiving rhGh treatment (SHORT + T) and control children (NORMAL) expressed per unit lean body mass. Individual values are shown with bar, indicating mean value for group SHORT + T significantly different from NORMAL $p < 0.05$.



5.4 DISCUSSION

The purpose of this study was to examine whether children of short stature consumed less food than children of normal stature and whether accelerated growth following treatment with rhGh in children of short stature was associated with changes in energy intake and metabolic demand. The energy demands of short stature children with or without rhGh treatment was also investigated since a previous study had indicated that BMR per unit LBM of short stature children was greater than that of children of normal stature [Walker 1990].

Although children of short stature who were not receiving treatment with rhGh consumed less energy in absolute amounts, than children of normal stature, when energy intakes are corrected for differences in body mass (either as body weight or as lean body mass), short stature children have comparable if not greater energy intakes. This challenges the expectation that being short is associated with a low energy intake and conforms with observations made by Walker in Jamaica on stunted children [Walker 1990]. The findings show that the combined effects of short stature and rhGh treatment result in a considerable increase in energy intake, whether expressed in absolute terms or per unit body mass, over and above that observed in children of either short or normal stature. Thus short children have relatively high energy intakes which are increased still further during rhGh treatment.

The BMR measures the metabolic demand for energy. In this study the shorter children had a higher BMR per unit body mass than taller children. Treatment with rhGh

increased the height of the short children but did not have any effect on BMR per unit body mass. The results suggest that the greater BMR cannot be explained simply by differences in height or in body composition. BMR expressed as a percentage of that predicted for age, sex and weight was within the normal range for both normal and short untreated children, but for the children treated with rhGh was 17% above that predicted. The treated children would be growing at a rate greater than normal and previous work shows that during rapid catch-up growth in children, BMR is related to the rate of weight gain [Brook 1974]. However, although a part (approximately 5%) of the increase in BMR following treatment with rhGh could be explained on this basis, the overall rate of growth is insufficient to account for the large relative increase in BMR. The greater BMR must therefore be associated with a difference in metabolic demand that is essentially an inherent characteristic of the shorter child: a characteristic that does not alter with the height spurt unlike anthropometric indices.

The results of the present study confirm the suggestion that rhGh treatment induces the deposition of lean tissue and mobilisation of adipose tissue. The metabolic demands of short children appear to be greater than their peers of normal height and these increased demands seem to be satisfied by an increased intake of food. The increased energy requirement appears to be a characteristic that remains inherent within the body and is not influenced by rapid growth engendered by rhGh treatment. Whether this increase in metabolic demand and intake persists during periods of normal growth following cessation of rhGh

treatment in short children remains to be seen.

CHAPTER 6

6. SUMMARY AND CONCLUSIONS

Assessing the adequacy of a child's diet is a fundamental challenge to a practising clinician or dietitian, particularly when faced with a child having disease or appearing to grow poorly. The standard approach is to compare the recorded derived nutrient intake against some reference point (such as DRV's) appropriate for a child of that age.

For example the Estimated Average Requirement (EAR) for a 9 year old boy is 8.55 MJ. This assumes, however, that the child is of an average height and mass for that given age. In practice this child may be very different in height, mass and body composition as a result of poor growth or disease.

This thesis addressed the issue of how dietary food patterns and nutrient intakes of children with short stature and those receiving growth hormone treatment differ from those children of normal height. Attention was directed to which foods provided the energy intake required and whether or not nutrient intakes met with current recommendations. BMR measurements were also taken and how the supply of energy consumed related to the metabolic demand.

The experimental data provides evidence that for body size short children consume an increased quantity of foods that are high in fat and refined carbohydrate, such as crisps, chips, sweets and sugar. These are the foods which provide the majority of the increase in energy intake in the diet of short children. When treated with growth hormone short children have a height spurt and tend towards more normal height. The food intake pattern which they exhibit

shows a further increase in the refined carbohydrate and fatty foods. The short children being treated with growth hormone consume at least twice as much confectionery and sugar as the normal height children. So far as the food patterns for meat, bread, cereals and milk are concerned, the percentage energy provided by these foods is very similar in all the groups when expressed in terms of body size. The vegetable and fruit intakes of all the short children were significantly lower. The short children whether receiving growth hormone treatment or not had less energy provided by vegetables and fruit compared to the normal height children.

So do short children selectively choose refined carbohydrate and fatty foods to eat or is it that they are members of a whole family who eat a similar diet and the child is just part of this ? It is not possible to say whether the short children have altered taste preferences for these foods and thus select them or whether it is the parents who provide the foods as a matter of course in the home environment. To a certain extent the pattern of food intake must relate to parental influence - parents buy and provide food for the family. Although it was not explored, the parents of the children in the study may have had financial constraints, poorer education, leading to the inability to buy and eat "healthy" foods such as fruit, vegetables and low fat/low sugar products.

As mentioned previously, the short children being treated with growth hormone have an increased percentage energy provided by refined carbohydrate and fatty foods, such as chips and confectionery. Whether this is related to the

growth hormone treatment altering appetite and taste preference in the child or as a result of the parents providing more of these foods, is a question remaining unanswered. The suggestion that the growth hormone in some way affects appetite and food choice must be further investigated.

For growth to occur, energy and other nutrients must be sufficient to meet metabolic demand. Body size needs to be considered when analysing a childs diet, since this will give an indication of the dietary quality. For example, the nutrient being considered in absolute amounts of mg or g per day, may be above or below the Dietary Reference Value when compared against age. This gives little indication whether for body weight the intake is sufficient to meet demand or indeed whether, for the amount of energy consumed, the nutrient intake is below or above the dietary reference standard.

The experimental section investigating nutrient intakes suggest that the pattern of nutrient intake of short stature children appears to differ relative to normal height children depending on how the intake is expressed. Nutrient intakes when expressed in absolute quantities of mg or g per day are decreased in the shorter children compared to the normal height children but still maintain on average at least 80% Reference Nutrient Intake (RNI). However when nutrient intake is expressed as relative proportionality (mg or g/MJ energy intake), both the short, normal height and growth hormone treated children consume similar nutrient intakes. This suggests that all the children have the same quality of diet irrespective of height. The extra food such

as confectionery and crisps that is being taken by the short children does not appear to lead to significant nutrient dilution and must contain nutrients in addition to the extra energy.

We have shown that the food patterns of the short and normal children are quite different, so one might expect the dietary quality of the childrens' diets to be different. It may be expected that a diet with an increased quantity of energy being provided by confectionery and snack foods has expected to have a lower nutrient intake, whether it be measured in absolute or relative proportionality values. Classically this has been expressed as the "empty calorie" hypothesis, a popular concept that has evolved. The experimental data in this thesis does not provide strong support for this concept. Some of the reasons for this could be due to the variability between groups, errors in the methods undertaken and that the sample size is too small to detect differences even if they do exist. It is not the only work that has cast doubt upon the "empty calorie" concept. Recently, a study by Gibson [Gibson 1993] has found that children consuming a diet with an increased quantity of refined carbohydrate have nutrient intakes that are similar to other children who are consuming less refined carbohydrate.

Nutrient intakes can be reflected by dietary food patterns. For example, the short children in this study have a percentage energy provided by vegetables and fruit that is decreased in comparison to the normal height children. One would expect that because of this, the dietary intakes of folate would be decreased in the diets that contain less

vegetable and fruit. The folic acid measurements in absolute amounts of µg/day is significantly lower in the short children compared to the normal height children. The same trend is seen when folic acid intake is measured per MJ of energy consumed.

When discussing dietary reference standards of Reference Nutrient Intake (RNI) and Lower Reference Nutrient Intake (LRNI) estimations of whether intakes achieve these standards can be calculated by knowing the total energy intake. The table over (Table 6.1) illustrates the quantity of energy required in order to achieve the RNI and LRNI for particular nutrients. The nutrient density in units/MJ in Table 6.1 is the mean value taken from all three groups of children. It can be seen that for the RNI of folic acid to be achieved, 9803kJ energy is required in the diet. This energy intake is by far increased when compared to the mean intake of the children, 7360kJ. The RNI of Vitamin B_1 and Vitamin C is reached by far less energy, approximately one third that required for folic acid. Table 6.1 illustrates that the LRNIs are achieved by a decreased quantity of energy, for example, folic acid requires only 4901kJ to reach the LRNI compared to 9803kJ needed for the RNI.

If the EAR for a 9 year old is taken, 8550kJ, then the iron, zinc and folic acid intakes of these children will not satisfy the dietary reference standards of the RNI. To satisfy the RNI, proportionately more energy needs to be consumed by the children.

Other factors which might relate directly to stunting are calcium, zinc, iron and Vitamin B_2. The data from this study illustrates that the increased energy intake seen in the

short treated children, 8133kJ, compared to the short untreated children,6228kJ does not alter the relative proportionality of the nutrients as might be expected. For example, the calcium intake expressed as mg/MJ is 81mg/MJ in the short untreated children but despite the increase of 1900 kJ/d observed in the short treated children, the calcium intake when expressed qualitatively is only 78mg/MJ.

Presently, there is a risk that short children being assessed against standard reference values for age, are asked to eat more food when in fact they are already consuming the same if not more, in terms of body size than a child who is taller. In the clinical environment, body size and ultimately metabolic demand should be known to assess nutritional status effectively. The childs' demand for energy has to satisfy the metabolism of the already existing tissues in the body and for the synthesis of new tissues. To what extent energy intake meets the metabolic demand of synthesizing new tissue will decide how well a child will grow and ultimately attain their final height. The experimental chapter investigating energy intakes and basal metabolic rates reveal subtle differences when energy intake and basal metabolic rate is measured either in absolute amounts or as an expression of body size. This thesis demonstrates that short children consume and expend less energy that the normal height child when expressed in absolute amounts. When expressed per kilogramme of lean body mass the short children then consume and expend an increased quantity of energy. Work in adults has also demonstrated similar findings. Metabolic demand is increased in individuals who are short in stature through chronic

Table 6.1 Quantity of energy required to achieve the Reference Nutrient Intake (RNI) and Lower Reference Nutrient Intake (LRNI) in a childs diet.

Nutrient	Mean nutrient density units/MJ[1]	RNI for children aged 7–10 years	kJ required to satisfy the RNI of nutrient	LRNI for children aged 7–10 years	kJ required to satisfy the LRNI of nutrient
Energy	7360 kJ				
Sodium	0.313 mg	1200 mg	3833	350 mg	1118
Potassium	0.303 mg	2000 mg	6600	950 mg	3135
Calcium	0.087 mg	550 mg	6321	325 mg	3735
Magnesium	0.026 mg	200 mg	7692	115 mg	4423
Iron	$1x10^{-3}$ mg	8.7 mg	8700	4.7 mg	4700
Copper	$1.4x10^{-4}$ mg	0.7 mg	5000	no LRNI	-
Zinc	$7.7x10^{-4}$ mg	7.0 mg	9090	4.0 mg	5194
Vitamin A	0.08 µg	500 µg	6203	250 µg	3101
Vitamin B_1	$1.5x10^{-4}$ mg	0.4 mg	2666	0.23 mg	1533
Vitamin B_2	$2.0x10^{-4}$ mg	1.0 mg	5000	0.5 mg	2500
Nicotinic Acid	$1.88x10^{-4}$ mg	6.6 mg	3510	4.4 mg	2340
Vitamin C	0.012 mg	30 mg	2500	8 mg	625
Vitamin B_{12}	$3.0x10^{-4}$ mg	1 µg	3333	0.6 µg	2000
Folic Acid	$1.5x10^{-2}$ µg	150 µg	9803	75 µg	4901

[1]Mean nutrient density observed in the diets of all three groups of children.

undernutrition when expressed per kilogramme of lean body mass [Soares 1991]. When considering the Energy Intake/Basal Metabolic Rate ratio, the short and normal height children have similar values. This suggests that the energy intakes in short children who are otherwise normal are sufficient to meet their requirement for metabolic demand.

As a child develops and increases in body size, the metabolic demand for energy per kilogramme of body weight decreases. This is due to an increased amount of somatic tissue effectively diluting the visceral tissue, organs such as the brain and heart. Visceral tissue has an increased metabolic demand compared to somatic tissue. Metabolic demand per unit mass is therefore greater in children who are small and short with a greater proportion of metabolically active tissue.

By using age related reference standards for estimating absolute energy intake, short children are at risk of being asked to eat more - more than they may require. For example a 9-10 year old boy of average weight, 30kg requires 8.55MJ/day. A 9-10 year old boy who is short will have a body mass nearer 22kg. Taking this weight, the child is nearer the average weight of a 6 year old, whose estimated energy requirements are 7.57MJ/day. This therefore highlights the need for practising clinicians and dietitians to use body size, anthropometric indices of height and body mass in their dietary assessments and recommendations.

Recently, a group of children have been matched for body size rather than age to the short 9-10 year old children who participated in this study [Wootton 1993, personal communication]. Various parameters were measured and are

illustrated in Table 6.2. The data presented is with non-parametric statistics as that is how the data of the normal height children is presented.

Table 6.2 Characteristics of normal height children aged 6 years and short children aged 9 years.

	Normal Children (N) 9 ♂ + 5 ♀ median (range)	**Short Children (S) 9 ♂ + 6 ♀ median (range)**	**Statistical significance S v N p - values**
Age (years)	6.17 (4.61 - 6.94)	9.10 (8.28 - 10.40)	$p < 0.01$
Height (cms)	117.1 (103.3 - 121.0)	117.0 (113.4 - 123.0)	NS
Height (SDS)	0.19 (-0.27 - 0.57)	-2.36 (-3.15 - -1.81)	$p < 0.01$
Body weight (kg)	20.8 (16.7 - 23.9)	21.3 (17.2 - 27.2)	NS
% body fat	14.9 (7.3 - 21.5)	16.5 (8.3 - 25.0)	NS
LBM (kg)	18.1 (13.6 - 20.4)	17.5 (14.5 - 21.4)	NS
BMR (kJ/d)	4260 (3560 - 4950)	4140 (3480 - 4680)	NS
BMR/LBM (kJ/kg/d)	243.4 (208.7 - 304.5)	227.2 (177.3 - 291.9)	NS

N = normal children (Wootton communication)
S = short children of present study
p values calculated with Mann-Witney test.

Quite clearly the normal height 6 year olds and short stature 9 year olds have similar height, body weight, lean body mass and Basal Metabolic Rates. This suggests that the increased metabolic demands seen in the short stature children are a function of body size rather than biological age.

At the moment health professionals focus more on the age of a child rather than on their body mass or height during

dietary assessment. The DRV's need to emphasize the discrepancies that can arise when only using age as a reference point.

When short children are treated with growth hormone, body height and body size increase. One would therefore expect metabolic demand to decrease but this was not observed. The short children on growth hormone treatment remain with an increased metabolic demand that has if anything been further increased. Growth hormone does have an anabolic and lipolytic effect on the body, whereby the short child becomes taller, leaner and more muscular. Anthropometrically these children then become more like normal height children but their metabolic characteristics remain similar to those of short children.

This observation would suggest that metabolic demand is a characteristic that is intrinsic. For example, a child anthropometrically and visually can alter with treatment of growth hormone but the characteristic of an increased metabolic demand for energy remains within them.

The treatment of a short child with growth hormone creates more difficulty in finding an appropriate reference standard to assess the adequacy of energy intake. These children prior to treatment are short in stature but have grown taller. The children however are not necessarily proportionally heavier and so body mass measurement may be inappropriate to use as a reference standard. Age will also be inappropriate to use as these children are still smaller than average for age. Therefore children on growth hormone treatment are a category of children who have no real suitable reference standard against which to assess energy

intake.

The results of this study are based on the assumption that the estimation of energy and nutrient intake, body composition and BMR are valid. For the measurement of energy and nutrient intake, a common methodology was employed in the three groups of children. Since the same method was used in each group, any errors that may have occurred will be equally applied to each group. Meticulous care was taken with the dietary assessments knowing that errors could occur. The energy and nutrient intakes observed in this study are consistent with the DRV's and other work [DHSS 1989]. There was no evidence that one particular group either over or under reported their food intake.

The values of body composition seen in this study are consistent with other workers [Gregory 1991]. For this study the author assumed the error of LBM measurements were consistent between and within the groups. The increased energy intake per unit LBM or BMR per unit LBM seen in the short stature groups are real and not simply the product of an underestimation. Such an error would mean that the short stature children would have an increased percentage body fat than they already have, to give rise to an underestimate of LBM. There is no indication that this was the case in this study. The same differences are apparent even if energy and BMR are expressed per unit body weight and not LBM.

No alternative methods of measurement were available at the time of this study that would measure the body composition of children more accurately.

Validation of Basal Metabolic Rate in children is

difficult to do. The BMR is the amount of energy required by the Lean Body Mass. Growth rates in children can alter with seasonal changes [Marshall 1971] and thus the amount of Lean Body Mass will change. Errors may also occur with the use of equipment. Datex have validated their equipment (Deltatrac) and providing certain precautions are adhered to then measurements are reproducible [Datex 1993]. Recent work has suggested that there is a 5% variation on measuring Basal Metabolic Rate in children [Bond 1992].

This thesis addressed, as a cross sectional study, the issue of how food patterns, nutrient intakes and metabolic demands of children with short stature and those receiving growth hormone treatment differ from those children of normal height. Further investigation needs to be directed towards a longitudinal study of these children seeing how food patterns, nutrient intake and metabolic demands alter as the children grow older.

Further studies with these children are required after their growth hormone treatment has been completed. The knowledge of whether the metabolic demand of these children may stay the same or alter is as yet unknown.

CONCLUSIONS

Short children have different food patterns to normal height children. They consume an increased percentage of energy from confectionery, crisps, and chips when compared to normal height children. This same food pattern is exhibited when short children are treated with growth hormone.

Energy intakes when expressed per kg of body weight are

increased in short children and short children on growth hormone treatment when compared to normal height children. The increased energy intake in these short children is fuelled largely by refined carbohydrate and high fat foods. Despite the difference in energy intake observed, the relative proportionality of the nutrients in all the childrens' diets are very similar.

The basal metabolic rates of short children and short children being treated with growth hormone are increased compared to normal height children, when expressed per kg body weight. Short children satisfy their metabolic demand for energy which is increased when compared to normal height children.

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Appendix

The following are abbreviations used in the Appendix tables.

case no. (1-15)	=	Short treated children (tables A.1-A.3)
case no. (16-30)	=	Short stature children (tables A.4-A.6)
case no. (31-40)	=	Normal height children (tables A.7-A.9)
Dec. age	=	Decimal age of child (years)
Ht.	=	Height (cms)
Ht. SDS	=	Height Standard Deviation Score
Wt.	=	Weight (kg)
Bic.	=	Biceps skinfold measurement (mm)
Tri.	=	Triceps skinfold measurement (mm)
Subs	=	Subscapular skinfold measurement (mm)
Supr	=	Suprailiac skinfold measurement (mm)
LBM	=	Lean Body Mass (kg)
%BF	=	Percentage Body Fat (%)
BMR	=	Basal Metabolic Rate (kJ)
Pred BMR	=	Predicted Basal Metabolic Rate (kJ)
Ener In.	=	Energy Intake (kJ/d)
Prot	=	Protein intake (g/d)
Fat	=	Fat intake (g/d)
CHO	=	Carbohydrate intake (g/d)
Na	=	Sodium intake (mg/d)
K	=	Potassium intake (mg/d)
Ca	=	Calcium intake (mg/d)
Mg	=	Magnesium intake (mg/d)
Fe	=	Iron intake (mg/d)
Cu	=	Copper intake (µg/d)
Zn	=	Zinc intake (mg/d)

Vit A	=	Vitamin A intake (µg retinol equivalent/d)
Vit D	=	Vitamin D intake (µg/d)
Vit E	=	Vitamin E intake (mg/d)
Vit B_1	=	Vitamin B_1 (Thiamin) intake (mg/d)
Vit B_2	=	Vitamin B_2 (Riboflavin) intake (mg/d)
Nico Acid	=	Nicotinic Acid intake (mg/d)
Vit C	=	Vitamin C intake (mg/d)
Vit B_6	=	Vitamin B_6 intake (mg/d)
Vit B_{12}	=	Vitamin B_{12} intake (µg/d)
Folic Acid	=	Folic Acid intake (µg/d)
Meat	=	Energy contribution of meat and meat products (kJ/d)
Milk	=	Energy contribution of milk and milk products (kJ/d)
Cake	=	Energy contribution of cakes and biscuits (kJ/d)
Conf	=	Energy contribution of confectionery, sugar, sweets and jam (kJ/d)
Chip	=	Energy contribution of chips and crisps (kJ/d)
Cer.	=	Energy contribution of cereals and bread (kJ/d)
Veg.	=	Energy contribution of vegetables and fruit (kJ/d)
Sugar	=	Sugar intake (Monosacharides and Disacharides) (g/d)
Starch	=	Starch intake (Starch and dextrins) (g/d)

Table A.1 Body composition, energy and protein intake in short treated children.

	Dec. Age	Ht.	Ht. SDS	Wt.	Bic.	Tri.	Subs	Supr	LBM	%BF	BMR	Pred BMR	Ener In.	Prot
1	9.9	127.9	-1.29	22.8	3.3	6.3	5.3	4.2	19.9	12.6	5070	4031	7382	47.9
2	9.9	126.2	-1.64	25.4	3.1	4.3	5.0	3.4	22.7	10.4	5250	4504	8946	59.7
3	9.3	125.5	-1.13	27.6	4.7	8.2	7.6	5.3	23.5	14.7	5410	4358	7428	43.8
4	9.5	128.1	-1.02	23.1	3.1	5.2	4.6	3.3	19.0	16.9	5130	4326	7389	49.5
5	9.9	127.7	-1.36	28.6	5.6	8.0	5.9	5.9	24.4	14.3	5210	4443	6804	52.8
6	9.0	122.6	-1.55	22.2	3.6	6.3	6.0	4.5	19.0	14.3	4790	4223	7181	38.9
7	8.2	116.9	-1.80	19.5	2.5	4.2	4.7	3.1	17.6	9.6	4480	3966	6933	47.8
8	9.0	121.2	-1.62	22.0	4.0	6.4	4.7	3.5	20.3	8.3	4650	3934	7641	42.0
9	9.1	120.5	-1.13	22.2	2.7	4.9	4.5	3.9	19.7	10.8	4730	4209	9329	53.4
10	10.1	130.1	-1.18	28.5	2.0	4.2	3.6	2.9	26.3	7.0	5150	4841	8291	49.8
11	8.5	119.4	-1.62	23.2	3.6	5.4	4.8	3.7	20.7	10.6	4680	4285	7011	44.1
12	9.2	120.5	-1.85	24.5	6.0	9.5	5.4	7.0	20.4	16.5	4930	4104	10233	63.8
13	8.9	119.9	-1.80	23.6	3.4	7.3	5.6	3.7	21.2	10.0	4530	4034	8784	50.0
14	8.8	117.4	-2.22	22.2	2.4	5.4	4.6	3.6	19.7	10.8	4456	4192	8569	53.1
15	10.4	130.8	-1.19	29.8	9.1	5.0	6.2	5.6	24.9	17.8	5460	4456	10826	56.3

Table A.2 Macro and micro nutrient, vitamin intakes in short treated children.

	Fat	CHO	Na	K	Ca	Mg	Fe	Cu	Zn	Vit A	Vit D	Vit E	Vit B_1	Vit B_2
1	77.9	230	2308	2196	691	186	7.6	1.05	6.20	485	1.73	5.16	0.96	1.23
2	83.4	302	2593	2997	832	323	11.4	1.80	9.36	1103	1.09	4.75	1.25	1.79
3	56.5	286	1788	2087	752	196	7.9	0.99	5.60	532	1.70	5.40	1.55	1.58
4	63.5	262	1803	2434	874	248	9.2	1.08	6.90	511	0.98	4.18	0.99	1.79
5	54.8	213	4717	2200	678	232	15.6	1.20	4.90	286	2.78	4.45	0.67	0.92
6	47.9	296	2163	1607	615	145	5.8	0.82	4.22	227	0.41	1.87	1.49	1.52
7	61.6	240	2077	2152	600	195	9.1	1.25	6.10	834	1.00	3.20	1.22	1.81
8	73.6	262	1553	2317	493	173	6.7	0.78	4.53	442	0.84	7.42	0.76	1.19
9	69.7	364	2450	2310	592	197	9.3	1.25	5.85	1798	1.41	4.50	1.83	1.77
10	76.2	288	1964	2049	542	173	8.3	1.14	5.53	397	1.75	4.38	0.79	0.62
11	63.9	243	1917	2515	317	201	7.9	1.09	5.04	389	1.00	4.64	1.06	0.89
12	103.0	333	2850	2599	796	268	12.9	2.40	8.90	2634	0.76	5.40	1.08	1.84
13	57.5	362	2256	2572	556	204	9.4	1.09	6.11	348	3.18	5.60	1.63	1.40
14	83.1	286	2213	2934	587	246	10.0	1.29	2.17	735	1.53	4.60	1.32	1.33
15	136.3	301	3574	3216	587	240	10.2	1.41	5.16	853	3.86	7.04	0.90	0.80

Table A.3 Vitamin and food group intake in short treated children.

	Nico Acid	Vit C	Vit B_6	Vit B_{12}	Folic Acid	Meat	Milk	Cake	Conf	Chip	Cer.	Veg.	Sugar	Starch
1	10.1	25	0.91	1.80	103.1	546	740	866	1426	981	1217	331	99	92
2	15.9	219	1.08	3.40	124.9	925	1087	699	1765	847	1309	524	153	105
3	13.9	257	1.18	1.97	123.5	202	703	655	1900	658	1011	198	154	103
4	12.3	201	0.95	2.32	100.6	554	1585	995	1250	729	1216	90	128	97
5	11.3	22	0.78	2.18	97.3	628	613	849	742	562	1563	384	60	108
6	15.5	166	0.51	1.25	93.3	330	692	1515	1710	499	1777	163	163	104
7	16.2	27	1.00	5.43	103.9	1206	613	1561	1307	428	922	700	126	86
8	13.3	33	1.13	1.38	95.1	542	429	1447	1887	1868	707	123	141	95
9	23.0	177	0.97	1.53	117.0	634	597	2109	1891	596	1958	659	168	168
10	10.8	167	0.81	1.08	112.2	654	441	799	2713	963	1210	411	171	95
11	13.4	62	1.15	1.17	112.8	484	23	1448	1167	1347	1102	620	105	102
12	16.4	29	1.20	9.6	188.0	1037	684	2621	2248	1057	1548	488	168	136
13	21.7	543	1.06	2.6	111.3	777	362	1087	2259	945	1633	553	212	116
14	15.6	120	1.12	2.78	125.0	1328	570	1525	1521	1470	1257	494	126	99
15	12.8	40	1.44	1.28	123.4	490	298	1428	1941	2451	1522	1518	111	57

Table A.4 Body composition, energy and protein intake in short stature children.

	Dec. Age	Ht.	Ht. SDS	Wt.	Bic.	Tri.	Subs	Supr	LBM	%BF	BMR	Pred BMR	Ener In.	Prot
16	9.7	117.4	-2.91	21.7	7.9	12.8	6.1	5.5	17.5	19.1	3480	4071	7218	59.7
17	9.3	114.9	-2.94	22.2	6.0	12.1	5.2	5.2	18.5	16.5	4140	3900	5863	42.8
18	10.0	123.0	-2.22	26.3	7.6	17.8	6.3	7.5	20.3	22.7	3600	4153	6790	43.6
19	9.6	118.0	-2.78	20.8	3.3	10.3	5.3	4.3	17.3	16.5	3720	4083	4687	31.5
20	8.5	111.4	-2.16	21.7	6.1	8.9	5.7	4.1	18.0	17.3	4220	4119	6977	48.1
21	8.4	117.0	-1.81	26.2	8.5	16.0	9.4	9.8	19.6	25.0	4680	4205	5761	33.1
22	8.5	117.6	-1.97	19.5	2.8	6.3	4.3	3.8	17.2	11.8	4090	3788	5817	36.2
23	9.7	122.0	-2.17	27.2	4.93	14.4	6.3	4.9	21.4	20.9	4460	4630	5449	33.8
24	9.1	117.7	-2.27	19.4	5.6	9.5	6.6	5.9	16.2	16.1	3520	3722	6541	38.5
25	8.9	115.8	-2.67	23.4	4.0	11.9	5.0	4.3	19.4	17.0	4250	4280	9039	70.0
26	8.9	115.2	-2.69	17.6	4.0	6.7	5.9	5.1	14.9	15.2	4350	3804	5739	35.0
27	9.1	113.4	-3.15	17.2	4.4	8.5	4.4	4.0	14.5	15.2	3890	3761	5859	32.7
28	8.3	115.2	-2.16	17.7	3.0	6.2	4.4	2.9	15.7	11.3	3820	3813	6120	36.6
29	9.3	117.0	-2.54	21.3	3.3	6.7	3.9	3.5	19.5	8.3	4430	3857	6376	33.8
30	8.7	115.9	-2.36	19.0	2.5	5.6	4.3	3.9	16.6	12.6	4500	3640	5179	28.1

Table A.5 Macro and micro nutrient, vitamin intake in short stature children.

	Fat	CHO	Na	K	Ca	Mg	Fe	Cu	Zn	Vit A	Vit D	Vit E	Vit B_1	Vit B_2
16	80.5	201	3137	2415	622	186	9.9	1.23	8.6	1011	0.70	4.73	0.80	1.17
17	51.0	202	1849	1709	515	135	6.3	0.73	4.5	632	1.65	4.00	0.78	1.00
18	58.1	243	1935	2077	636	186	7.8	1.00	5.9	571	0.85	3.62	0.90	1.00
19	53.8	134	1748	984	392	90	3.9	0.47	3.1	196	0.91	3.07	1.27	1.23
20	60.7	244	2101	2358	807	192	7.4	0.89	5.2	223	1.12	3.56	1.01	1.61
21	53.9	200	1963	1505	441	144	5.9	0.72	3.8	123	0.59	4.80	1.08	0.87
22	45.6	219	1859	1681	738	165	4.7	0.79	3.9	609	1.86	2.65	1.18	1.55
23	62.7	159	2742	1518	373	124	6.8	0.79	3.9	854	2.84	4.62	0.62	0.99
24	65.5	216	1149	1739	394	171	7.2	0.97	5.3	229	0.94	4.03	0.52	0.73
25	77.8	308	2350	3195	486	252	11.2	1.60	5.2	734	3.30	6.68	1.13	0.67
26	58.4	186	1557	1772	421	171	6.5	0.99	4.1	180	1.50	4.17	1.05	0.92
27	52.5	210	1669	1850	546	162	6.5	0.86	3.8	248	0.25	2.82	1.13	1.26
28	60.5	203	1592	1953	357	133	5.7	0.86	4.2	144	0.29	3.50	0.80	0.74
29	52.0	242	1244	1801	495	142	5.8	0.83	3.6	266	0.59	2.60	0.74	0.89
30	50.8	176	1444	1408	355	141	6.5	0.85	3.4	220	1.64	3.85	0.66	0.72

Table A.6 Vitamin and food group intake in short stature children.

	Nico Acid	Vit C	Vit B_6	Vit B_{12}	Folic Acid	Meat	Milk	Cake	Conf	Chip	Cer.	Veg.	Sugar	Starch
16	12.7	45	1.00	3.40	125.7	1383	699	134	910	716	1195	496	66	94
17	11.8	190	0.74	2.05	87.6	614	388	1414	1020	369	1077	311	92	91
18	11.3	136	0.81	1.41	131.5	386	889	1244	833	606	992	530	136	83
19	13.7	17	0.45	0.87	44.1	723	466	360	407	590	649	19	29	87
20	11.4	103	0.80	1.54	148.9	549	787	781	1507	1128	955	7	111	85
21	12.8	24	0.82	0.58	77.2	173	320	735	883	1408	2129	34	56	133
22	12.5	21	0.59	1.50	68.4	133	133	268	1362	425	1526	342	101	89
23	9.0	15	0.62	2.17	105.1	840	840	0	590	783	1428	226	37	92
24	8.4	15	0.82	1.85	64.3	694	694	1021	1767	856	869	459	108	82
25	13.7	121	1.55	2.74	179.0	347	347	1811	505	807	1506	817	140	135
26	11.7	70	1.05	2.89	107.7	497	497	446	1413	757	949	466	89	79
27	12.2	94	0.64	1.13	81.7	279	280	147	1614	1087	1408	157	91	82
28	10.0	20	0.83	0.85	62.7	622	623	1137	1622	1252	435	370	102	73
29	9.62	128	0.67	1.35	61.5	342	632	1236	1880	715	644	470	144	74
30	7.39	44	0.55	0.90	70.0	246	235	558	1703	686	849	181	90	72

Table A.7 Body composition, energy and protein intake in normal height children.

	Dec. Age	Ht.	Ht. SDS	Wt.	Bic.	Tri.	Subs	Supr	LBM	%BF	BMR	Pred BMR	Ener In.	Prot
31	9.6	127.1	-.132	26.7	6.8	4.4	4.8	3.2	23.1	13.4	5010	4616	8184	74.6
32	9.7	139.5	0.80	25.6	9.1	6.0	4.2	3.2	22.3	12.6	4870	4309	8205	64.3
33	9.7	142.4	1.28	28.4	11.8	9.8	6.4	10.5	22.0	22.3	5430	4712	6873	44.6
34	8.9	132.3	0.17	23.7	4.3	2.8	4.2	3.2	20.7	12.6	4748	4397	8253	70.8
35	9.9	145.1	1.42	35.7	9.8	5.6	5.7	7.0	29.0	19.1	6810	5452	9079	52.6
36	10.3	135.2	-0.43	27.7	10.5	7.0	6.0	5.8	22.9	16.9	4380	4436	6002	52.8
37	9.6	134.7	0.03	28.5	10.8	8.8	7.0	6.0	23.0	19.1	4770	4480	6896	48.8
38	9.8	139.1	0.52	29.9	9.6	6.9	4.4	2.9	24.8	17.0	4970	4944	9670	74.7
39	9.2	139.9	0.47	40.2	11.5	20.0	15.5	11.0	27.9	30.6	5010	4969	5945	44.6
40	8.6	135.1	0.92	27.7	8.0	5.8	4.8	5.2	22.9	16.9	5160	4741	9491	62.8
41	10.5	139.2	-0.08	33.7	12.0	9.5	9.0	8.5	24.9	25.9	5660	5237	8442	51.4
42	10.0	133.4	-0.55	32.0	10.0	14.0	7.2	6.9	24.4	23.7	4120	4548	6718	46.0
43	10.0	140.1	0.48	32.6	13.0	7.5	6.1	6.2	25.6	21.4	5740	5170	7900	62.4
44	9.6	133.5	-0.08	29.8	8.0	9.8	6.0	4.2	24.7	16.9	5300	4569	7950	58.8
45	8.7	130.1	-0.02	29.2	10.2	8.0	7.4	9.0	22.8	21.8	5400	4837	8051	55.2
46	10.5	141.2	0.27	50.0	24.0	13.7	15.0	15.0	32.0	35.2	5690	6366	5842	39.9

Table A.8 Macro and micro nutrient, vitamin intake in normal height children.

	Fat	CHO	Na	K	Ca	Mg	Fe	Cu	Zn	Vit A	Vit D	Vit E	Vit B_1	Vit B_2
31	76.0	256	2823	3621	1209	275	10.8	1.60	8.7	629	1.53	4.48	1.70	2.24
32	92.6	230	2165	3360	448	254	10.1	1.40	7.4	567	1.00	7.11	1.29	0.92
33	68.7	223	1825	1847	830	174	8.1	0.89	6.5	522	1.67	3.46	0.87	1.00
34	76.3	264	2167	2779	700	280	10.9	1.42	9.00	604	1.75	3.87	1.28	1.31
35	72.5	343	2694	1814	780	203	7.1	1.18	5.4	296	1.36	2.20	1.23	1.56
36	55.5	190	2404	2115	447	154	7.6	1.01	6.2	581	1.36	3.31	1.09	1.26
37	68.8	219	3002	2771	474	243	9.1	1.37	7.8	1020	1.14	4.67	1.46	1.73
38	53.2	401	4313	3087	1542	316	8.1	1.15	7.6	728	2.68	4.00	4.98	5.49
39	61.2	182	1644	1810	882	166	6.3	0.95	5.5	582	2.20	4.04	0.56	1.23
40	92.9	311	2910	2523	1056	238	9.7	1.18	7.2	846	0.90	4.30	1.47	1.82
41	66.7	315	2497	1857	852	223	9.5	1.08	6.2	395	0.66	3.46	1.30	1.97
42	56.1	240	3085	2302	591	194	7.4	1.02	5.6	226	1.35	3.94	2.18	1.27
43	73.0	258	2567	2457	964	207	9.3	1.14	7.5	1606	2.11	5.11	0.75	1.43
44	85.2	234	3252	2621	592	214	8.9	1.25	8.3	878	0.69	4.49	1.58	1.96
45	76.1	268	2437	2132	570	204	10.2	1.14	7.4	732	2.85	5.17	0.99	1.26
46	57.3	190	1659	2336	706	175	6.1	0.89	4.8	440	0.56	4.19	1.16	1.30

Table A.9 Vitamin and food group intake in normal height children.

	Nico Acid	Vit C	Vit B_6	Vit B_{12}	Folic Acid	Meat	Milk	Cake	Conf	Chip	Cer.	Veg.	Sugar	Starch
31	15.9	153	1.46	2.94	286.0	97	243	9	153	70	168	132	120	94
32	17.0	135	1.74	1.46	213.0	1323	510	393	1819	1695	417	1160	131	87
33	7.2	160	0.65	1.50	97.7	558	1009	998	821	305	1234	362	72	72
34	14.7	69	1.20	2.80	156.3	1029	1080	1947	668	398	1569	1327	112	122
35	15.4	23	0.73	1.79	81.7	661	994	821	3513	418	1858	419	213	107
36	15.2	41	0.77	3.67	93.3	1359	380	1182	302	646	1141	630	62	94
37	19.0	40	1.23	5.48	159.4	1216	513	276	609	1136	1453	1371	59	94
38	52.0	143	1.16	3.01	168.1	18	1983	1056	692	127	4135	732	106	243
39	5.9	190	0.68	2.29	89.0	480	1003	738	1204	944	472	765	95	54
40	17.2	49	1.02	1.80	131.9	628	1261	1680	1419	712	1535	420	145	115
41	16.5	146	0.78	1.60	136.1	377	1044	1476	1812	626	2221	107	150	143
42	10.3	54	0.82	1.22	138.9	536	533	1011	1117	861	1313	432	119	92
43	11.3	21	1.07	3.04	105.1	1069	808	1946	621	517	1592	621	106	125
44	19.3	34	1.10	2.54	128.0	1259	1071	681	409	756	1935	409	72	121
45	14.3	82	1.91	4.07	111.1	1494	424	982	1397	0	1869	1397	110	126
46	12.1	43	1.02	1.27	116.9	115	709	601	582	271	1065	582	55	105